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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 57 MAY, 1947 NO. 5

THE PNEUMOENCEPHALOGRAM OF CEREBELLAR ATROPHY*

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CHICAGO, ILLINOIS

THE roentgenographic appearance of the cerebellum is often neglected in descriptions of pneumoencephalograms or described in terms of the size of the fourth ventricle and cisterna magna only. Careful study of air-patterns in the posterior fossa, according to Davidoff and Dyke,[2] may reveal the following:

In the anteroposterior view, the gas in the subarachnoid space outlining the cerebellum forms an inverted V, the limbs of which are supported by the quadrangular lobes of the hemispheres and the apex of which covers the culmen of the vermis. The upper portion of the fourth ventricle is usually visualized as a triangle. Laterally, virtually the entire cerebellum may be seen in profile, surrounded by the cisterna ambiens anteriorly, subtentorial air dorsally and caudally, and the cisterna magna posteroinferiorly. The superior vermis and its individual components (lingula, lobulus centralis, culmen, and declive) may be identified. Posteriorly, the quadrangular lobules of the hemispheres can sometimes be recognized in the normal. Air is often seen as thin lines between contiguous folia. The tonsils are not uncommonly defined by gas in the cisterna magna.

Detailed inspection of the encephalographic appearance of the contents of the posterior fossa is of greatest interest where degenerative disease or maldevelopment of the cerebellum is suspected, and objective confirmation of clinical impressions may be obtained thereby. In addition, air-study of the brain, particularly in lateral views with the occipital region of the head superior, can reveal unexpected cerebellar disease. The pneumoencephalogram thereby provides a means of substantiating (but not necessarily rejecting) the diagnosis of cerebellar atrophy and may bring to attention unsuspected degeneration of the cerebellum as a primary process or complication of other known disease of the nervous system.

Some of the cases to be presented (of non-neoplastic cerebellar disease) were those in which encephalography confirmed the clinical diagnosis of cerebellar degeneration, but in others air studies revealed undiagnosed involvement of the cerebellum. Exact correlation of the roentgenographic appearance of the hindbrain with symptomatology could not be made in all cases manifesting clinical evidence of cerebellar disease. This is not, however, surprising considering the great difficulty experienced in attempting to differentiate the various

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types of cerebellar atrophy even under the microscope.

Structural abnormalities of the cerebellum may or may not be associated with cerebellar symptomatology[8] but, if the abnormalities are of sufficient magnitude, they may be visualized by pneumoencephalography. In this report clinical and roentgenographic studies of a variety of cases in which air studies revealed gross defects of the cerebellum are presented.

Anatomical alterations of the cerebellum may be divided into two categories—the atrophies and the malformations. The term "atrophy," although it merely signifies a diminution in size, is nevertheless always the manifestation of a diseased state and should not be spoken of as primary.[6] From a gross examination of the brain it is often impossible to decide whether one is dealing with an atrophy or a malformation and detailed microscopic study is often necessary and, as noted, may still not produce the final answer.

Cerebellar atrophies may be further subdivided as follows:[7]

A. Progressive degeneration (so-called "primary"), due to hereditary causes, intoxications, inflammatory processes, or unknown cause.

B. Degeneration of the cerebellum secondary to disease of the pontile and olivary nuclei (olivopontocerebellar atrophy), disease of the contralateral cerebral hemisphere ("crossed atrophy"), vascular disease.

C. Progressive cerebellar degeneration complicating Friedreich's ataxia, bulbar paralysis, peroneal muscular atrophy, epilepsy, feeblemindedness, and so forth.

Cerebellar malformations may likewise be considered under several headings:

A. Aplasia (complete)
B. Partial aplasia
C. Hypoplasia
D. Dysplasia.

The above outline is included for purposes of orientation and clarity. However, the cases described below do not offer illustrative examples of each of the multiplex variants of cerebellar atrophy and maldevelopment. The more common types of cerebellar disease (non-neoplastic) encountered in clinical neurology are included, with more than one instance of several classificatory diagnoses.

In all cases, the technique of encephalography was standard and closely similar to that recommended by Davidoff and Dyke.[2]

REPORT OF CASES

Primary degeneration or secondary (olivopontocerebellar) atrophy of the cerebellum (class A or B in above outline).

In the following 4 cases, the appearance of the cerebellum in the pneumoencephalogram confirmed the clinical impression of cerebellar atrophy. Roentgen examination did not, however, offer a solution as to the differential diagnosis between olivopontocerebellar atrophy and parenchymatous cortical atrophy (Parker and Kernohan[11]). The cases are listed under the heading of "primary" or secondary cerebellar degeneration to distinguish them from the other members of this series and also because an accurate pathogenetic diagnosis could not be made.

Evidences of cerebellar degeneration were most striking roentgenographically and consisted principally in extreme widening of interfolial sulci and accentuation of demarcation of the subdivisions of the vermis and hemispheres.

CASE 1. R. P. (Hosp. No. 313481), a white male, aged forty-four, was admitted to the Illinois Research Hospital on January 25, 1946, because of difficulty in walking, dysarthria, and generalized weakness for the prior one year. The patient had been a drug addict (morphine and heroin) in the past, but had broken himself of the habit. The gait was extremely ataxic, on a broad base. The patient swayed and fell in the standing position with the eyes open or closed. Speech was dysarthric and scanning. There was no true nystagmus. All of the usual cerebellar tests of force, range, and direction of movement were positive. The deep reflexes were symmetrically exaggerated, the abdominal reflexes were sluggish, Hoffman reactions were present, and there was ankle clonus. Gastric analysis (hista-

mine) was negative for free hydrochloric acid. The remainder of the routine laboratory examinations, including serology of blood and spinal fluid, were negative.

In the lateral view of the encephalogram (Fig. 1) there was striking decrease in the size of the whole cerebellum. The cisterna magna was greatly increased in size, and the fourth ventricle was somewhat enlarged. The sulci demarcating the semilunar lobules from the declive were especially broad and deep, as was the superior posterior sulcus. The lobulus centralis culmen and declive were small. In the anteroposterior view the posterior fossa seemed to be almost entirely filled with air. Where seen, the

Fig. 1. Case 1. R. P. Parenchymatous cortical cerebellar atrophy or olivopontocerebellar degeneration. Shrinkage of components of vermis cerebelli, especially of lobulus centralis and declive.

surfaces of the hemispheres were nodular (Fig. 2).

The clinical diagnosis was olivopontocerebellar atrophy, although the history of drug addiction and gastric achlorhydria suggested parenchymatous cortical atrophy.[11]

Case II. S. W., a white male, aged forty-five, was successively admitted to Gallinger Municipal Hospital, Washington, D. C., on May 14, 1939, September 25, 1940, August 22, 1941 and November 22, 1941. The first hospital admission was necessitated because of a suicidal attempt. The only significant physical findings were a self-inflicted laceration of the neck and severe deafness, said to have existed for two years. The patient had been heavily alcoholic in the past. He recovered from a reactive depression

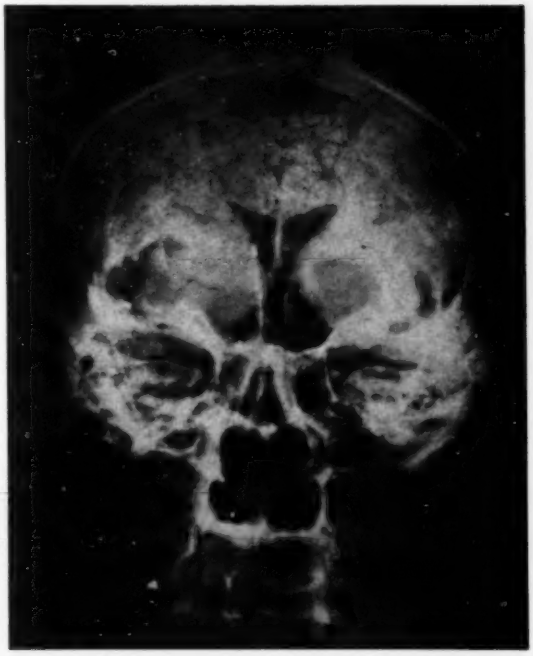

Fig. 2. Case 1. R. P. Anteroposterior view of encephalogram. Posterior fossa almost entirely filled with air due to extreme atrophy of cerebellum.

Fig. 3. Case II. S. W. Olivopontocerebellar atrophy or "primary" degeneration. Tremendous enlargement of cisterna magna, nodulation of surface of atrophic cerebellum. Hydrocephalus ex vacuo. Lateral view.

and was discharged, to be readmitted on October 25, 1940, because of progression in deafness, and the appearance of ataxic gait. Examination disclosed severe disturbance in

FIG. 4. Case II. S. W. Posterior fossa filled with air. Cerebellar atrophy, anteroposterior view.

walking, decomposition of voluntary movements, hyperactive deep reflexes, absence of superficial reflexes, and nerve-type deafness bilaterally. Lumbar puncture obtained xanthochromic fluid with a cell count of 5,000 erythrocytes and a total protein content of 1,700 mg. per 100 cc. Blood and spinal fluid Wassermann reactions were negative.

In the lateral encephalogram, hydrocephalus ex vacuo and cerebral cortical atrophy were evident (Fig. 3). The cisterna magna was enormous and coalesced with air over the posterosuperior aspect of the cerebellum. The cerebellum was reduced to small size, with scalloping and nodulation of the surfaces of the hemispheres and profile of the vermis. In the anteroposterior view (Fig. 4) only the lateral extremities of the hemispheres projected into the air-filled posterior fossa.

The clinical and encephalographic diagnoses were olivopontocerebellar atrophy, far-advanced, or "primary" atrophy of the cerebellum due to alcoholism or causes unknown.

CASE III. M. H. (Hosp. No. 99446), a white female, aged fifty-five, was admitted to the Illinois Research Hospital on May 9, 1945, because of vertigo, ataxic gait and dysarthria of two years' duration. The patient was unable to walk because of extreme ataxia, speech was scanning, there was coarse nystagmus on lateral gaze, positive cerebellar signs, and generalized hypotonia. The usual laboratory examinations, including serological, were negative.

Pneumoencephalography disclosed increased air in the posterior fossa (Fig. 5). The cisterna magna, however, did not appear to be increased in size. The lingula, lobulus centralis, culmen and declive were well outlined. The semilunar lobules were easily identifiable and appeared almost transparent superiorly. The most striking feature of the appearance of the cerebellum was widening of the clefts between the major subdivisions of the vermis and hemispheres.

The clinical and roentgenographic diagnosis was cerebellar atrophy (olivopontocerebellar atrophy?).

CASE IV. V. J. (Hosp. No. 104020), a colored female, aged thirty-one, was admitted to the Illinois Research Hospital on February 26, 1946, with a history of progressive loss of balance and generalized weakness for the prior eleven years. Physical examination disclosed ataxic gait, vertical and horizontal nystagmus, slurring speech, dysmetria and dyssynergia, hyperactive deep reflexes and positive Hoffman reac-

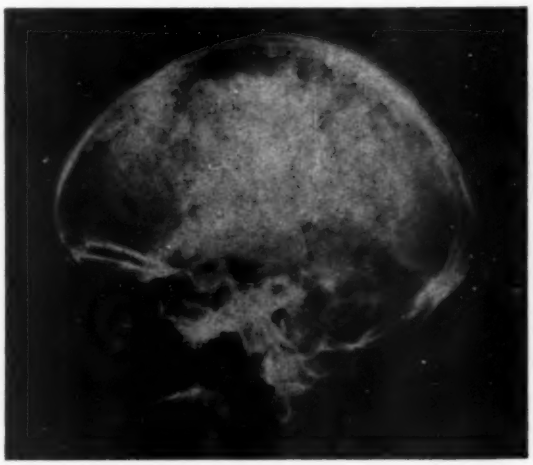

FIG. 5. Case III. M. H. Cerebellar atrophy (olivopontocerebellar atrophy?). Enlargement of fissures demarcating lingula, lobulus centralis, culmen and declive.

tions. All laboratory examinations, including serology, were negative save for a mid-zonal colloidal gold curve in the spinal fluid.

In the lateral encephalogram (Fig. 6), the cisterna magna and fourth ventricle were only slightly, if at all, increased in size. There was a moderate decrease in volume of the whole cerebellum. The anterosuperior and posterosuperior sulci were unusually prominent, and atrophy accentuated the interfolial fissures. There was slight internal hydrocephalus and cerebral cortical atrophy as well.

The clinical and encephalographic diagnoses were cerebellar atrophy, either parenchymatous cortical or olivopontocerebellar in type.

Cerebellar atrophy secondary to or associated with cerebral atrophy.

Atrophy of the cerebellum as a part of general brain atrophy is of not uncommon occurrence and, in reviewing the encephalograms of several cases of diffuse cerebral atrophy, varying degrees of degeneration of the hindbrain were seen. In one case, of unknown etiology (possibly Pick's disease) and complicated by a history and physical findings suggesting occlusion of the posterior inferior cerebellar artery, cerebellar atrophy was so striking that it merits presentation.

Case v. J. S. (Hosp. No. 101501), a white male, aged thirty-two, was admitted to the Illinois Research Hospital on September 14, 1945, with the chief complaint of loss of vigor and

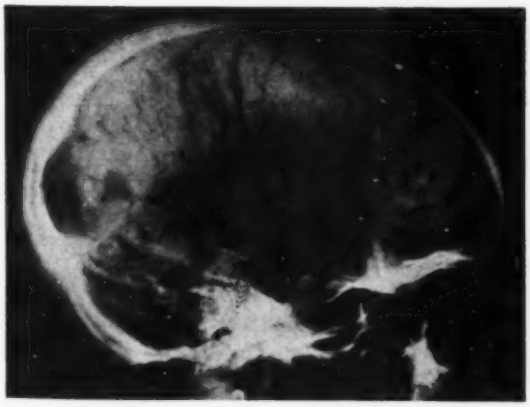

Fig. 6. Case iv. V. J. Parenchymatous cortical or olivopontocerebellar atrophy. Atrophic accentuation of interfolial fissures of cerebellar hemispheres. Cerebral cortical degeneration.

Fig. 7. Case v. J. S. Possible Pick's disease. Fluid levels in posterior fossa, enlargement of cisterna magna. Outline and demarcation of components of vermis well marked.

ambition for the previous one year. Two years before admission the patient was said to have suffered a "stroke" which left him with a right hemiparesis and left hemisensory defect. There had also been hypersomnolence and irritability. Diminished visual acuity was present in the right eye; there was nystagmus on right lateral gaze, a right abducens paresis, sensory loss in the trigeminal distribution on the right, right spastic hemiparesis, and loss of superficial sensibility on the left side of the body below the face. The protein content of the spinal fluid was 62 mg. per 100 cc. Laboratory examinations, including serology, were otherwise normal.

Air studies revealed entirely unsuspected diffuse cerebral atrophy of far-advanced degree, maximal in the temporal lobes (Fig. 7), with accompanying hydrocephalus ex vacuo. In addition, cerebellar atrophy was clear cut. Components of the profile of the vermis were exceptionally well demarcated, the cisterna magna was enlarged and in the upright position (Fig. 7) fluid levels were present in the posterior fossa.

An elderly woman with mental deterioration and personality changes was found to exhibit clinical indications of associated cerebellar disease. Pneumoencephalography substantiating this diagnosis, revealing atrophy of the cerebellum of even greater magnitude than cerebral cortical degeneration.

Case vi. H. S. (Hosp. No. 105268), a white

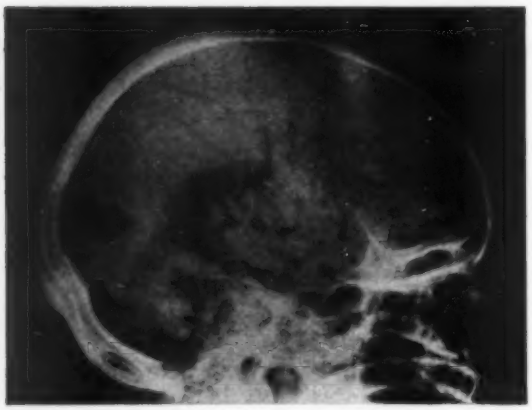

FIG. 8. Case VI. H. S. Cerebral and cerebellar atrophy. The cisterna magna is large and its anterior limits ill defined. Wide sulci in the vermis.

female, aged fifty-nine, was admitted to the Illinois Research Hospital on May 10, 1946. There had been difficulty in walking and progressive deterioration of mentality (loss of memory, confusion, euphoria) during the prior three years. She had been confined to a bed or wheel-chair for twelve months. Dysarthria had been noted for five months and urinary and fecal incontinence had developed shortly before admission. There were many evidences of early organic psychosis including inappropriate smiling and laughter. Nystagmus was present on lateral gaze. Incoordination was most pronounced in the legs. The deep reflexes were hyperactive. The patient was unable to sit or stand unassisted. There was no control of bowel or bladder. All of the usual laboratory examinations, including blood and spinal fluid serology, were normal.

Encephalography (Fig. 8) demonstrated a greatly enlarged cisterna magna and wide interfolial sulci in the vermis. There were signs of cerebral atrophy, particularly frontally.

Cerebellar atrophy secondary to traumatic encephalopathy.

Degeneration of the cerebellum is likewise known to accompany cerebral atrophy resulting from trauma. Encephalographic evidence of post-traumatic cerebellar atrophy has been presented by Lemere and Barnacle,[5] in whose case the cisterna magna and fourth ventricle were both extensively enlarged. In Case VII, atrophy is indicated only by cisternal expansion, but the latter is of a degree compatible only with degeneration of the cerebellum.

CASE VII. R. S. (Hosp. No. 82592), a white male, aged twenty, was admitted to the Illinois Research Hospital on April 27, 1942, because of grand mal epilepsy of two years' duration. The onset of convulsive seizures followed by one month a severe head injury, since which time he had also had a left-sided weakness. Examination disclosed a mild left hemiparesis including the face. Routine laboratory examinations were not unusual. There was no clinical suspicion of cerebellar disease.

Dilatation of the ventricular system, especially on the right, was shown in the encephalogram. As noted, the cisterna magna was large and full, in the absence of subdural air, and the cerebellum, therefore, reduced in size (Fig. 9).

CASE VIII. D. P. (Hosp. No. 106580), a white male, aged fifty-one, was admitted to the Illinois Research Hospital on August 2, 1946, in a state of severe mental confusion which had existed since head injury in an automobile accident two months before. He had remained unconscious for one week following the injury, and upon regaining consciousness had been restless, noisy, confused, and disoriented. He was transferred from a mental institution for study. The patient manifested the usual signs of organic psychosis, and in addition had a slight left hemiparesis and bilateral loss of superficial re-

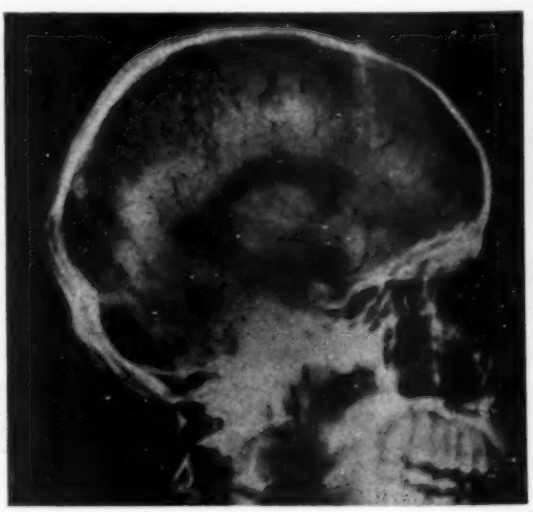

FIG. 9. Case VII. R. S. Traumatic encephalopathy. Enlargement of cisterna magna.

flexes. There was probably a left homonymous hemianopsia as well.

Spinal fluid examination was not remarkable in any respect, and roentgenograms of the skull did not reveal fracture. An electroencephalogram disclosed a slow wave focus in the right occipital area. Pneumoencephalographic study showed moderate hydrocephalus ex vacuo, encephalomalacic atrophy of the right occipital pole, and severe atrophy of the cerebellum, evidenced largely by gross enlargement of the cisterna magna, but also by dilatation of the fourth ventricle (Fig. 10).

Cerebellar degeneration complicating Friedreich's disease.

As Hassin[4] has reported, cerebellar atrophy may be associated with Friedreich's ataxia. The next 3 cases illustrate this condition. The pathological findings in the air studies were of variant type and degree. This may be related to the extent of necrobiosis or be due to fundamentally different anatomical changes in the individual cases.

Case ix. B. F., a white male, unmarried, aged twenty-four, was admitted to St. Elizabeth's Hospital, Washington, D. C., on February 13, 1942. The patient was one of four brothers, all of whom suffered from Friedreich's ataxia. Other siblings were normal, and there was no hereditary history of the condition. Onset of neurological symptoms was at the age of five years. The salient neurological findings were bilateral sixth nerve paresis, reduction of upward gaze, wild ataxia, positive cerebellar tests, weakness and atrophy of the peripheral musculature, and absence of the deep reflexes. The mental state was that of euphoric immaturity. All of the usual laboratory examinations were within normal limits.

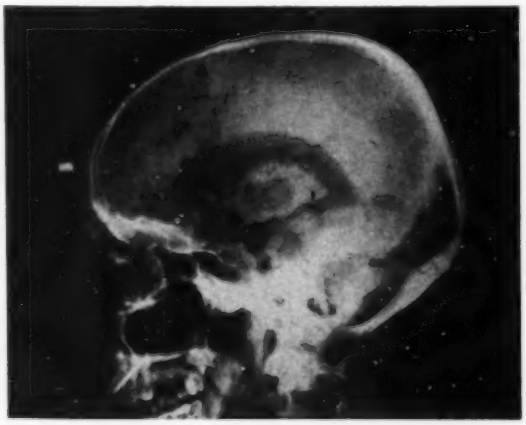

Fig. 10. Case viii. D. P. Traumatic cerebellar atrophy, with dilatation of cisterna magna and enlargement of fourth ventricle. Encephalomalacia of occipital pole.

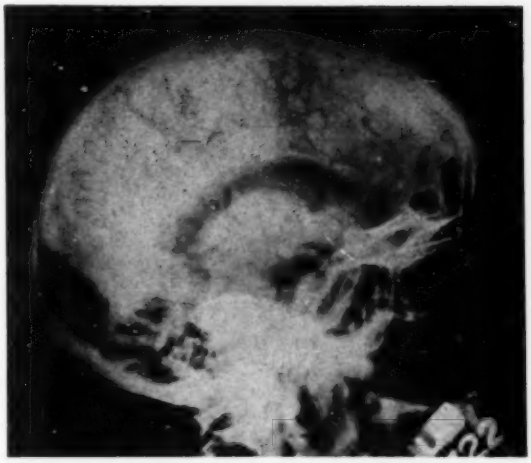

Fig. 11. Case ix. B. F. Friedreich's ataxia. Increase in size of interfolial sulci of the cerebellum with reduction in number of convolutions is apparent.

Encephalography (Fig. 11) revealed wide interfolial sulci, giving a finger-like appearance to the leaves of the cerebellar hemispheres. The encephalograms were submitted to Dr. Cornelius G. Dyke, of the Neurological Institute of New York, for examination, and his report stated: "The sulci outlining the cerebellar folia are entirely too large, which I believe is due to the atrophy of the cerebellum.... The atrophy of the cerebellum as seen in the encephalogram I believe is due to cerebellar sclerosis."

Case x. E. G. (Hosp. No. 102639), a white female, aged twenty-four, was admitted to the Illinois Research Hospital on November 27, 1945, because of staggering gait for the past seven years. One brother had Friedreich's ataxia. Ten years before admission paresthesias had appeared, and since the age of seventeen there had been inexorable progression of ataxia. Physical examination disclosed scoliosis, dysarthria, spastic paraplegia, absence of vibratory sensation below the ribs, and absence of deep reflexes. Laboratory examinations were normal. The clinical diagnosis was Friedreich's ataxia.

Moderate but definite cerebellar atrophy was

FIG. 12. Case x. E. G. Friedreich's ataxia. Moderate cerebellar atrophy, widening of sulci and fissures.

evident in the encephalogram (Fig. 12). This was expressed as widening of the sulci and fissures demarcating the semilunar lobules, declive, and culmen. The fourth ventricle was slightly larger than normal, but the cisterna magna was of average size.

CASE XI. H. L. (Hosp. No. 103672), a white male, aged twenty-four, was admitted to the Illinois Research Hospital on February 6, 1946, with a history of progressive ataxia of ten years' duration. A brother, aged twenty-seven, was admitted with the same difficulty and was also diagnosed as Friedreich's disease. Other siblings were unaffected and there was no hereditary history of this or other neurological conditions. Examination revealed kyphosis of the spine, nystagmus, dysarthria, decomposition of movement, absence of tendon reflexes, loss of vibratory sensation in the legs, and peripheral hypalgesia of glove and stocking type. Laboratory examinations including electroencephalography were not unusual. The diagnosis was Friedreich's disease.

Cerebellar atrophy was evidenced in the pneumoencephalogram by an unusually large cisterna magna (Fig. 13). The fourth ventricle was also increased in size. The air had not outlined the individual folia. Dilatation of one occipital horn was the only other roentgenographic abnormality.

Cerebellar malformation—hypoplasia.

In cerebral agenesis (more properly, dys- or hypogenesis) encephalography may disclose associated hypoplasia (or secondary atrophy?) of the cerebellum. Four such cases are included in this series.

CASE XII. J. M. (Hosp. No. 105858), a white male, aged twenty-three, was admitted to the Illinois Research Hospital on June 20, 1946, for evaluation of convulsive seizures which had responded poorly to medication. The patient had always been mentally retarded and had sustained several minor head injuries during convulsions. Physical examination disclosed an unusually large head, nystagmus in all directions of gaze, left hemiparesis with Babinski sign, and slight ataxia on the left. The usual laboratory examinations were not remarkable with the exception that the electroencephalogram revealed a right temporoparietal focus.

Pneumoencephalography showed slight hydrocephalus ex vacuo and unsuspected virtual absence of the cerebellum. In a lateral projection a few striations were all that could be seen in an air-filled posterior fossa, and in the anteroposterior view the subtentorial region seemed to be devoid of cerebellum (Fig. 14 and 15). The degree of involvement was such as to make the diagnostic impression one of severe cerebellar hypogenesis.

CASE XIII. C. H. (Hosp. No. 106510), a colored male, aged four months, was admitted

FIG. 13. Case XI. H. L. Friedreich's ataxia. Gross enlargement of cisterna magna and fourth ventricle.

to the Illinois Research Hospital on July 23, 1946, for treatment of right-sided jacksonian seizures which had been present since birth. Delivery had been accomplished with difficulty after a prolonged labor, and the infant was resuscitated slowly. Examination showed scaphocephaly, premature closure of the sutures, and nystagmus on lateral gaze. Right-sided clonic seizures were almost constant while the child was awake. Routine laboratory studies were negative.

Slight hydrocephalus was manifest in the pneumoencephalogram, which also disclosed an

Fig. 14. Case xii. J. M. Lateral encephalogram, revealing posterior fossa almost completely filled with air save for few striations indicating surfaces of hemispheres. Cerebellar hypogenesis.

air-filled posterior fossa in which no cerebellar components could be recognized (Fig. 16). The impression was that of aplasia or hypogenesis of the cerebellum.

Case xiv. R. de V. (Hosp. No. 84825), a white male, aged nineteen months, was admitted to the Illinois Research Hospital on September 3, 1942, because of left-sided convulsions and difficulty in walking of two months' duration. Physical examination revealed gross nystagmus on looking to the right, inability to stand or walk, and falling to the right.

Symmetrical internal hydrocephalus was evident in the pneumoencephalogram (Fig. 17). The fourth ventricle was larger than normal

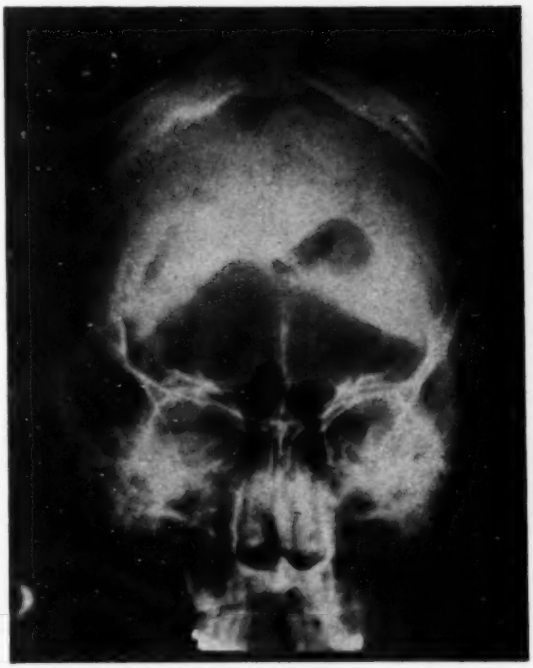

Fig. 15. Case xii. J. M. Anteroposterior encephalogram. Cerebellum not visible, subtentorial region filled with gas.

and a large coalescence of air filled the cisterna magna. The whole cerebellum was smaller than normal.

Case xv. B. S., a white male, aged two, was admitted to the Illinois Research Hospital on

Fig. 16. Case xiii. C. H. Cerebellar aplasia or hypogenesis. Space below tentorium apparently filled completely with air; no cerebellar components identifiable.

Fig. 17. Case xiv. R. de V. Cerebellar hypoplasia (atrophy?) accompanying maldevelopment of cerebrum. Cisterna magna and fourth ventricle increased in size as compared with normal. Hydrocephalus.

December 1, 1942, because of right hemiparesis and expressive aphasia noted since the age of eight months. Examination disclosed a profound spastic hemiparesis on the right and paucity of vocabulary.

The lateral ventricles were enlarged and the third and fourth ventricles were especially well outlined due to increase in size. The cisterna magna was enormous (Fig. 18).

Cerebellar hypoplasia (atrophy ?) was not suspected clinically in any of these patients although retrospective analysis of the Case xiv might suggest this diagnosis on the basis of the physical findings. The encephalographic features of cerebellar involvement consisted of enlargement of the cisterna magna and fourth ventricle, the folia not being demonstrated in any of the cases since they were probably not shrunken away from each other so as to widen the sulci as they are widened in atrophy.

DISCUSSION

Enlargements of the cisterna magna and fourth ventricle in the encephalogram have been emphasized as indications of cerebellar atrophy in previous reports on this subject.[1,3,5,9,10] It would also be anticipated that an extensive degree of degeneration of the hindbrain would result in widening and deepening of lines of air normally demarcating the subdivisions of the vermis and hemispheres. This conclusive evidence of cerebellar cortical atrophy was most striking in our cases of parenchymatous cerebellar cortical degeneration or olivopontocerebellar atrophy (which we are loath to attempt to differentiate) and in Friedreich's disease.

Correlation of the degree of local cerebellar involvement with clinical symptomatology and physical findings is not, we believe, possible from consideration of the encephalographic patterns. The latter are necessarily somewhat limited in definitive significance because of the almost invariable failure of injected air to completely delimit every cavity, cistern, and other extension of the subarachnoid spaces.

It is important to re-emphasize the declaration of Lemere and Barnacle[5] that air located above the cerebellum (subtentorial gas) cannot be interpreted as evidence of cerebellar atrophy and that in cases of true atrophy an increased amount of air lies posterior to the cerebellum, principally in an enlarged cisterna magna. An important

Fig. 18. Case xv. B. S. Cerebellar hypoplasia (atrophy?) associated with cerebral dysgenesis. Cisterna magna extremely large. Hydrocephalus.

observation noted in consideration of the air studies described is that whereas dilatation of the cisterna magna and enlargement of the fourth ventricle are found in both atrophy and hypoplasia, widening and deepening of clefts and fissures demarcating components of the vermis and hemispheres from each other and, in particular, increase in width of interfolial sulci were found only in cases of extensive atrophy of the cerebellum, either a primary or secondary degenerative process. The late growth of the folia of the cerebellum may account for the absence of deep fissuring of the surfaces of the organ in cerebellar hypoplasia, as may the developmental character of malformation of the hindbrain.

SUMMARY

Fifteen cases in which there was pneumoencephalographic evidence of cerebellar atrophy or degeneration or hypoplasia are presented. Cerebellar involvement in these patients was "primary" (?), the result of pontomedullary nuclear degeneration, cerebral atrophy, trauma, associated with Friedreich's disease, or the cerebellum was hypoplastic in a condition of cerebral dysgenesis.

Cerebellar atrophy may appear as enlargement of the cisterna magna and fourth ventricle, or as widening and deepening of interfolial sulci and fissures, or as a combination of these abnormalities. Scalloping and deep fissuring of the surfaces of the hemispheres and nodulation of the vermis are especially evident in cerebellar degeneration of the parenchymatous cortical or olivopontocerebellar types and in Friedreich's disease. Increase in size of the cisterna magna and fourth ventricle as compared with the normal is a feature of cerebellar hypoplasia. This may be related to initiation of the pathological process at the start of the developmental period or to the immature form of the cerebellum in infancy.

Case II is included in this series through the kindness of Dr. Walter Freeman of Washington, D. C.

Case VII was reported previously (Murphy, J. P. Atypical Friedreich's ataxia. *Yale J. Biol. & Med.*, 1943, *15*, 603–609).

Miss Roxana Pickering and Mr. Willard Huntzinger lent technical assistance in the preparation of this report.

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HEMANGIOMA OF VERTEBRA ASSOCIATED WITH SPINAL CORD COMPRESSION*

CASE REPORT

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HEMANGIOMAS of the vertebrae present themselves for clinical consideration in one of four ways: (1) Asymptomatic vertebral hemangioma, discovered incidentally in the course of roentgen examination of the chest, genitourinary tract, gastrointestinal tract, and so forth. These are unaccompanied by subjective symptoms or objective neurologic abnormalities, and are commonly managed by periodic observation only. (2) Symptomatic vertebral hemangioma with localized spinal and/or radicular pain, without objective signs of damage to the nerve roots or spinal cord, usually discovered on roentgen examination of the spine for the complaint of back pain. There is agreement that this situation is best treated by roentgen therapy with fairly successful therapeutic results. (3) Vertebral hemangioma accompanied by the subjective symptoms and objective signs of compression of the spinal cord, the myelopathy being due to associated epidural hemangioma. Types 1, 2, and 3 are characterized by preservation of the normal contour of the vertebral body; our case report and discussion will concern itself with an example of type 3 and the controversy which exists concerning its treatment. (4) Hemangioma of the vertebra which has undergone pathological fracture and loss of the normal contour of the vertebral body, with or without accompanying compression myelopathy. This rare type is usually diagnosed only by pathologic examination.

Because of the scarcity of reported observations of the third type which have been treated exclusively with roentgen therapy and the disagreement over the best methods of treatment, the following case report is presented.

CASE REPORT

G. W., a white male physician, aged fifty-eight, was admitted to Percy Jones General Hospital on May 9, 1945, because of inability to walk. In March, 1945, the patient first noted moderate ataxia in both lower extremities, followed shortly by mild weakness in both lower extremities, nocturnal pains in the spine between the scapulae, minimal difficulty in initiating micturition and numbness ascending from feet to umbilicus. All of the symptoms progressed slowly until one month prior to entrance at which time the unsteadiness and weakness of the lower extremities prevented walking without assistance. He denied neurologic symptoms prior to March, 1945, although a lumbar puncture had been performed for unexplained reasons three years previously during the course of a routine physical examination. It is presumed that some asymptomatic physical sign of spinal cord disease was present at this time.

Examination on admission failed to reveal any abnormalities of the heart, lungs, abdomen or genitals; the blood pressure was 130/80. There were rare pinhead-sized hemangiomas over various portions of the skin, none in the distribution of the upper dorsal cord segments. Examination of the cranial nerves was negative, and strength, tone, power, coordination and the deep tendon reflexes were normal in both upper extremities. With the assistance of two persons the patient was able to walk with a markedly spastic ataxic gait; muscle tone was moderately increased in both lower extremities, accompanied by a moderate, diffuse, symmetrical paresis in both lower extremities. The abdominal reflexes were absent in all four quadrants, the cremasteric reflexes were absent, the knee jerks were bilaterally hyperactive, more markedly so on the left than on the right, and there was abortive left patellar clonus. The Achilles reflexes were relatively diminished; there was no ankle clonus, but there were bilateral Babinski and Chaddock signs. The sense of position was well preserved in the toes, vibra-

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tory sensation was diminished below the eighth dorsal spinous process, pain sensation was impaired below the fourth dorsal cord segment level, tactile sensation was impaired below the fifth dorsal cord segment level, and temperature sensation showed no sharp upper level of disturbance, but was impaired below the seventh dorsal cord segments bilaterally. Inspection and palpation of the spine was negative, and there was no involuntary dorsal paravertebral muscle spasm. There was mild tenderness to thumping over the fourth, fifth and sixth dorsal spinous processes.

The blood hemoglobin, white blood cell count, sedimentation rate, urinalysis, serology and roentgen examinations of the chest, genitourinary tract and gastrointestinal tract were normal. Examination of the cerebrospinal fluid revealed an initial pressure of 160 mm. of water, and a complete manometric block on the Queckenstedt maneuver. The spinal fluid was clear, colorless, and contained no cells; the Wassermann reaction was negative, the colloidal gold curve ooooooooo, and the total protein content was 116 mg. per 100 cc. Roentgen examination of the spine (Fig. 1 and 2) showed alternating linear striations along the sinusoidal system of the fourth dorsal vertebral body, and loss of the normal homogeneous structure of the vertebral body, with extension of the mottled

Fig. 1. Hemangioma of fourth dorsal vertebral body. In the anteroposterior view the vertebral body is obviously increased in density, being considerably more radiopaque than other thoracic segments. In this projection a bubble-like appearance of the bone trabeculae is observed with no evident angulation or compression of the vertebral body. Thickened trabeculae, shown at *a* in the lateral view, are apparently sufficiently strong to prevent collapse of the centrum. In our original roentgenograms the heads and necks of the third and fourth ribs displayed irregular bone rarefactive changes indicating the presence of hemangiomatous elements, outside the spinal axis. Compare vertebral body at *a* with supra- and subjacent vertebrae.

FIG. 2. Enlargement showing detail of fourth thoracic hemangioma. The posterior processes are involved. Note the cystic changes due to angioma in the head and neck of the right third rib at *a*.

appearance into the laminae and proximal portions of the ribs bilaterally. The normal contour of the vertebral body was well preserved, and there was no disturbance in the adjacent intervertebral disc spaces. Pantopaque myelography (Fig. 3 and 4) revealed a complete block at the level of the lower margin of the body of the fourth dorsal vertebra.

Beginning on May 12, 1945, and continuing until June 2, 1945, a total of 2,700 roentgens was administered to the patient through a 10 by 15 cm. field centered over the fourth thoracic vertebra.

Two and one-half weeks after the start of roentgen therapy, definite subjective and objective improvement had commenced, and a physiotherapy and walking exercise program was instituted for the patient. At the end of three months the interscapular pain had entirely disappeared, the sphincter disturbance had returned to normal, there had been a substantial improvement in motor power and coordination of the lower extremities, and the patient was able to walk for three or four blocks without assistance before fatigue would necessitate resting. The sensory impairment over the trunk and lower extremities had shown substantial subjective improvement. Examination at this time revealed a mild Brown-Sequard syndrome at the fourth dorsal cord segment level with impairment of sensation over the left half of the trunk, and a mild degree of spastic paresis in the right lower extremity, of lesser degree than was previously present. A lumbar puncture performed on August 16, 1945, revealed an incomplete Queckenstedt block with a delayed rise and fall on jugular compression; the total protein content of the spinal fluid had fallen to 91 mg. per 100 cc. The myelogram was repeated at this time, with the finding that a few drops of oil passed the previously complete block at the fourth dorsal vertebral level, although there was still a definite obstruction.

Further roentgen therapy was then given and the patient continued to show slow progressive improvement. When last examined on October 10, 1945, five months after the beginning of treatment, there was still mild numbness and tingling in the legs, minimal ataxia particularly after dark, and the patient was able to walk for several miles without fatigue or disability. No disturbance in motor power or muscle tone was demonstrable in either lower extremity, and there was only the faintest degree of incoordination on the finer tests of equilibrium in the lower extremities. There was a vague impair-

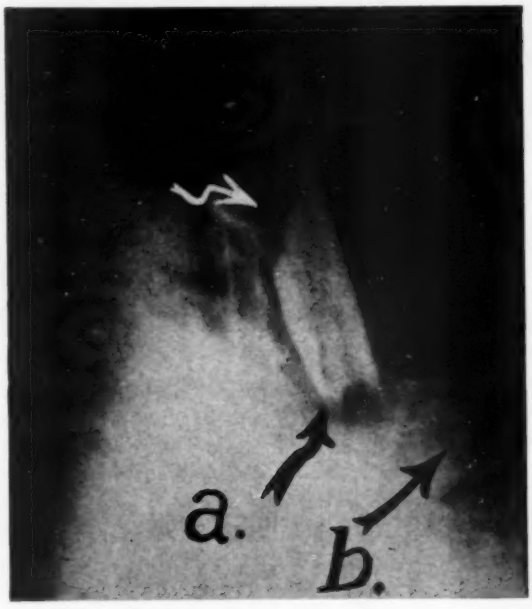

FIG. 3. Appearance of extradural hemangioma. Complete block of pantopaque column is shown at *a*. The lucid band in central portion of pantopaque column presents the outline of the spinal cord. The characteristic increased density of fourth dorsal vertebral body with irregular striations approximately three times normal width is shown at *b*. Patient in head down position. Lateral projection.

Fig. 4. Spot roentgenograms of pantopaque column—patient in prone Trendelenburg position, showing complete block at *a* and feathered appearance of pantopaque column along its cephalic margin. The appearance is identical with that of the first case described by Bucy and Capp. Following deep roentgen therapy another myelographic study revealed that the complete block had been converted into an incomplete obstruction. However, no definite alteration in the appearance of the trabeculae was observed. Improved appearance of the vertebral body and pedicles with sharper definition of the striations has been reported by others following such treatment.

ment of all modalities of sensation over the lower abdomen and lower extremities with no definite upper border which could be delineated. The abdominal reflexes were equal and active in all four quadrants, the cremasteric reflexes were absent, the knee jerks were of normal activity, the Achilles reflexes absent, and there were bilateral Babinski signs. The patient reported by letter that improvement was still continuing nine months after the institution of treatment.

TECHNICAL CONSIDERATIONS

In the last decade roentgen therapy of vertebral lesions, as well as tumors compressing the spinal cord, has improved with development of machines capable of producing reasonably high kilovoltages, on the order of 200–400 kv., and requiring somewhat heavier filtration than in use heretofore. In consequence, larger tumor doses can be attained with relatively greater safety and with less skin damage. By judicious timing of the courses of treatment, normal structures are spared, while neoplastic tissue can be made to regress or otherwise influenced to the benefit of the patient. The technique employed in this hospital is similar to that described by Pendergrass:[7]

1. Our objective is to deliver 3,000 to 4,000 r (with backscatter) into the center of the tumor.

2. Physical factors: 220 kv., 50 cm. target-skin distance, 0.5 mm. Cu and 1 mm. aluminum filtration. Half-value layer

1.43 mm. Cu. Output 43 r per minute.

3. Fields: (a) One posterior 10 by 15 cm. field accurately localized to the lesion by combined roentgenography and roentgenoscopy.

(b) Two anterior fields measuring 10 by 15 cm., placed in the parasternal position in order to cross-fire the fourth dorsal vertebral body and adjacent vertebrae from the anterior aspect of the chest wall.

The first series was directed entirely through the important single dorsal field, between May 12 and June 2, 1945. Since small initial doses are the rule in this clinic in order to obviate any untoward increase in intraspinal pressure, the first day the dorsal field received 50 r (measured in air). The second day 100 r was given. Since no unusual reaction followed, daily treatments of 200 r were then administered for a total dose of 2,700 r, with but a single rest period between May 24 and 31. The time consumed was fifteen treatment days. At this point a definite epidermitis sicca was observed.

Between August 6 and 29, 1945, a second series was given, using two 10 by 15 parasternal fields in addition to the above described dorsal field. In nine treatment days the posterior field received an additional 400 r (air), while 1,000 r was delivered to each of the two parasternal ports.

Between October 8 and 9, 1945, an additional 400 r was delivered to the dorsal field, making a total of 3,500 r; the total dose delivered to both anterior fields was 2,000 r. The estimated tumor dose in the spinal canal at the fourth thoracic level was 3,526 r (backscatter included); the estimated dose in the center of the fourth dorsal vertebral body was 3,376 r (backscatter included). There was moderate pigmentation of the skin with no evidence of telangiectasis five months after therapy was begun. The patient's subjective and objective improvement was such that no further therapy appeared indicated.

DISCUSSION

The characteristic alternation of vertical densities in the fourth dorsal vertebral body, the transverse myelitis at the same level and the prompt improvement in myelitic symptoms and neurologic signs following roentgen therapy seem ample justification for the diagnosis of vertebral hemangioma, epidural hemangioma, and compression myelopathy of the upper dorsal spinal cord.

It has been argued that radiation therapy should not be the primary method of treatment in these cases because of the delay in the relief of compression of the cord. Bucy and Capp[1] state that "Roentgen therapy alone should never be used in these cases for the length of time required for obtaining satisfactory results is so long that irreparable damage to the spinal cord would result during the course of treatment." It is noteworthy that their patient received a total of 659 roentgens immediately following laminectomy prior to discharge from the hospital and had effected a complete recovery eight months after laminectomy and radiation therapy. Karshner, Rand and Reeves[4] state that "The use of roentgen therapy alone in cases of compression myelitis seems not only inadequate but hazardous, for irreparable damage to the cord may occur before any beneficial effects can be anticipated." Their case was treated by decompressive laminectomy and high voltage roentgen therapy instituted on the thirteenth postoperative day, with an excellent recovery eighty days after the institution of therapy. This erroneous notion that a prolonged latent period follows roentgen therapy before benefit is evident is not supported by the case of Nattrass and Ramage[6] where recovery began immediately after roentgen therapy was instituted, that of Michon, Grégoire and Lafont[5] where recovery began eight days after the institution of radiation therapy, the case of Ferber and Lampe[2] who had begun to improve thirteen days after the onset of therapy, or by our patient in whom probable improvement commenced on the tenth day and was quite definitely present by the seventeenth day after the

institution of the radiation treatment.

The objection to surgical treatment is the mortality rate of from 20 to 25 per cent due to uncontrollable bleeding when decompressive laminectomy is attempted. Ferber and Lampe, who summarized the literature and reported one case of their own with a very satisfactory result treated exclusively by roentgen therapy, state that of the patients surviving operation, 10.7 per cent are unimproved, 14.3 per cent show improvement and 46.4 per cent have recovered, with the outcome in 2 cases unknown. Of the patients treated by laminectomy one-third have received some form of irradiation after operation. Of 13 cases of vertebral hemangioma associated with compression of the cord in which radiation therapy was the sole method of treatment, 10 made a complete clinical recovery, 2 made an incomplete recovery, and in the third complete transverse myelitis occurred during the course of radiation therapy.[3] A three-stage laminectomy was then performed and with the combination of surgical decompression and roentgen therapy the patient had made a clinical recovery at the end of two years.

It is felt that in those patients in whom the diagnosis of hemangioma of the vertebra with a slowly advancing myelopathy can be established by clinical and roentgenologic methods, the initial treatment of choice is roentgen therapy. No mortality is associated with the method and only one adverse report of acute complete transverse myelitis developing during the course of irradiation has been found. It is conceded that acute transverse myelitis occurring in the course of compression myelopathy from vertebral and epidural hemangioma is best treated by an immediate decompressive laminectomy. It is not felt that the delay of seven to twenty days before signs of beginning release of spinal cord compression appear following roentgen irradiation seriously impairs the patient's degree of ultimate recovery in the usual type of slowly progressing myelopathy.

SUMMARY

A case of epidural hemangioma associated with vertebral hemangioma and compression myelopathy is presented, the patient showing an excellent clinical recovery following radiation therapy.

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DISSEMINATED GRANULOMA INGUINALE OF BONES

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GRANULOMA inguinale is generally considered a local disease affecting the mucous membranes and skin of the genital and inguinal regions. That on occasions it becomes widespread, extending to other parts of the body, is evident from the review of Mayer and de Rocha Lima[5] in 1927, and from the more recent paper by Pariser and Beerman,[8] as well as from isolated reports that in the past few years have appeared in increasing numbers. Its occurrence in bones was reported as long ago as 1903–1904 by Hoffmann,[3] but one must refer to the recent case reports for satisfactory descriptions and illustrations of the osseous manifestations. To the best of our belief, no report has as yet appeared in the roentgenological literature and we therefore wish to present another case report with a review of the important findings of the previous case reports illustrating the roentgenographic changes noted.

Paggi and Hull[6] reported the case of a female, Negro, aged twenty-one, with osteolytic lesions in the medial end of the left clavicle, the anterior portion of the right sixth rib, and the medial part of the right scapula. These lesions followed a primary lesion in the cervix uteri which had extended to the vulva. Donovan bodies were demonstrated in the ulcerated cervix and labium major, and likewise in the aspirated material obtained from the abscesses overlying the affected bones.

Lyford, Scott and Johnson[4] reported 3 cases in which osteolytic destruction of the bones occurred. In their first case, roentgenograms revealed diffuse lysis of all small bones of the hands and feet, areas of osteolysis in the left fibula, both tibiae at the distal ends, skull, both ulnae, the distal end of the right clavicle and acromion of the right scapula. The lesions were purely osteolytic, without residual sequestrum or evidence of surrounding osteoplasia. Destructive changes were noted in the bodies of the fourth and fifth lumbar vertebrae and left hip in their second case. Osteolytic changes of the left first metacarpal bone in their third case was followed by destructive changes of the carpal bones and distal end of the left radius. The progress of the disease necessitated excision of the first metacarpal, then a guillotine amputation of the left mid-forearm, and finally amputation at the surgical neck of the humerus. The lesions in the bones contained Donovan bodies, and at operation no pus was obtained. The cortex of the bone about the areas of friable granulation tissue was thinned.

Sheldon, Thebaut, Heyman and Wall[10] reported the case of a Negro, aged forty-three, who developed an ulcer of the left leg. Roentgenologically, a large osseous defect in the proximal tibia at the level of the tubercle was noted. This defect, about 4 cm. in diameter, extended from the anterior surface to the posterolateral aspect resulting in a dissolution of the cortex. The bone cavity was surrounded by moderate sclerosis with irregular periosteal thickening along the medial, lateral and posterior aspects of the proximal portion of the tibia. Moderate osteoporosis of the distal femur and tibial condyles existed. A cyst-like area of rarefaction about 1.0 by 1.5 cm. was noted in the anterior portion of the seventh rib on the left. This area was surrounded by a zone of sclerosis with no periosteal reaction. No other bones were involved. They isolated the Donovan bodies by cultivation from the granulation tissue from the leg.

Palik and Schenken[7] reported the case of a female, Negro, aged twenty-one, with a primary lesion of the cervix uteri giving rise to two metastatic lesions in the left

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clavicle associated with abscesses of the overlying soft tissue which later ulcerated; a metastatic lesion of the anterior portion of the right sixth rib, and one in the right scapula revealing a punched out area of decreased density along the upper vertebral border near its spine. The histopathological changes of the lesions were typical of granuloma inguinale and smears contained Donovan bodies.

REPORT OF CASE

A female, Negro, aged thirty-three, was admitted to the service of Dr. Vaux at the Pennsylvania Hospital on August 15, 1944, with the complaint of a foul bloody vaginal discharge, pain in the left hip and nausea of a month's duration. She gave a history of having had a supravaginal hysterectomy, bilateral salpingo-oophorectomy and appendectomy elsewhere in 1935. She had previously had four miscarriages occurring about the second month of gestation, and had received antisyphilitic treatment. The remainder of her past history was not pertinent. On physical examination she had low fever of 100.4°F. and a rapid pulse. Both inguinal regions were tender on palpation. No scars or external lesion of the vulva or inguinal region existed and none developed throughout her course of illness. The cervical stump of the uterus was almost completely distroyed by a soft ulcerating bleeding growth which involved the vaginal vault and was continuous with a mass in the left posterior pelvis. Two biopsies of the vaginal wall revealed vascular granulative tissue with acute and chronic inflammatory cells but the underlying specific lesion of granuloma inguinale was not recognized in the tissue until later. On September 22, 1944, the pelvic mass was explored through a sinus tract in the vagina, and a great deal of necrotic tissue but no pus was obtained. Considerable induration existed about the sinus tract. By proctoscopic, cystoscopic and barium enema examinations, the mass in the left posterior pelvis was found to be pressing upon the rectum, sigmoid colon and bladder; but no intrinsic lesions were observed in these organs. The left ureteral orifice could not be seen on cystoscopic examination and no dye appeared in this area after intravenous injection of indigo carmine. Intravenous urog-

FIG. 1. Right leg, anteroposterior and lateral views. Note areas of subperiosteal rarefaction.

FIG. 2. Left leg, anteroposterior and lateral views.

Fig. 3. Lateral view of lower portion of left leg to show areas of rarefaction in fibula.

raphy demonstrated a non-functioning left kidney. On the basis of these findings, a continued hectic fever, and continuously retrogressive course, the left kidney was removed. The kidney was enlarged, pale, almost white, with a little focal necrosis microscopically but with no extensive disease. A psoas abscess behind the kidney was drained.

Later, a third biopsy of the vaginal lesion revealed the characteristic specific granulomatous tissue consisting of many vacuolated macrophages filled with Donovan bodies, associated with scattered collections of polymorphonuclear cells, and a careful review of previous vaginal and cervical biopsies disclosed occasional macrophages with Donovan bodies, which could be identified only after a prolonged search.

On November 4, 1944, she first complained of a painful and tender swelling over the left temporomandibular region. Roentgenographic examination of this region revealed nothing and the swelling soon subsided, but shortly thereafter she complained of painful nodules over the forehead, the left radius, the left tibia and elsewhere. Roentgenographic examination of these regions disclosed the presence of osseous lesions in both tibiae, both fibulae (Fig. 1, 2, 3 and 4), both ulnae, the top of the left radius, the shaft of the proximal phalanx of the right ring finger, both frontal bones and the left astragalus. The entire shaft of each tibia was involved mainly along the anterior surface; the fibulae were involved in the distal thirds of their shafts; the left astragalus along the superior aspect just anterior to the articular surface with the tibia and fibula. There was a single osteolytic lesion of the lateral tip of the distal head of the left radius while both ulnae showed involvement of their distal halves (Fig. 5). The metastatic bony defects of the skull were localized in the frontal region. Most of them were

Fig. 4. Same as Figure 3 to show appearance in anteroposterior view of fibula.

clustered about the suture line between the two frontal bones but several occurred close to the left frontoparietal suture (Fig. 6). In several views of the skull, these could be seen to have invaded the diploic cancellous bone between the inner and outer tables.

There was a generalized demineralization of the bones of the extremities.

The defects in the bones were multiple, irregular, osteolytic lesions of the cortex with no evidence of sequestrum or surrounding osteoplasia. The margins of the lesions were indistinct, fading into normal appearing bone. They lacked the punched out appearance of the osteolytic lesions seen, for example, in multiple myeloma. There was no sign of periosteal proliferation or periostitis.

Aspiration of the tibial lesion and biopsy later revealed the characteristic cells of granuloma inguinale containing Donovan bodies. Tartar emetic and later fuadin were given but too late to be effective, and on December 12, 1944, she was found dead lying in a pool of blood that had escaped from the vagina.

The significant pathologic changes noted at necropsy were as follows: The body was emaciated. Two scars, the result of salpingo-oophorectomy and supravaginal hysterectomy years before, and that of the recent nephrectomy which still remained open about a small sinus in the left flank, were present. None about the inguinal or perineal region suggested the healed lesions of granuloma inguinale. Poorly developed blood clots and watery malodorous exudate escaped from the vagina. Adhesions separated the peritoneal viscera from the large fibrous seminecrotic mass which lay in the pelvis and extended on the left over the pelvic brim into the region of the left psoas muscle. Scattered ulcers with granulating and necrotic bases extended from the vagina to the cervical stump which was almost completely destroyed. Sinuses extended to the outside of the bladder and rectum. A few shallow ulcers were in the mucosa of the bladder and in the mucosa of the rectum where the greatest bulge existed outside the bowel. Dense inflammatory tissue involved the outside of the left ureter near the pelvic brim. The left hypogastric vein was eroded and from this the fatal hemorrhage extended by the vaginal sinus to the exterior. Irregular firm hemorrhagic masses were scattered in the lungs near the pleura, and these contained softened gray pink centers. A small cortical erosion existed in the left fibula, and from the abscess underneath thin gray pink pus escaped. In the lower right fibula was another small abscess about 1 cm. in diameter and here too the outer

Fig. 5. Left forearm. Note area of rarefaction.

Fig. 6. Lateral view of skull. Note abscess in frontoparietal area.

FIG. 7. Portion of skull removed to show punched out defect. Compare with Figures 6 and 8.

cortex had almost entirely disappeared. A similar small depressed oval defect lay in the outer table of the frontal bone (Fig. 7). Microscopic examination of these gross lesions revealed many endothelial macrophages, vesicular and frequently packed with the pale staining, almost inconspicuous Donovan bodies, associated with polymorphonuclear cells. Interspersed here and there were the large pathognomonic cells described by Pund and Greenblatt.[9] The final pathologic diagnosis was granuloma inguinale of the cervix uteri with ulceration of vagina, bladder, rectum and extension to retroperitoneal tissue about the pelvis, upward over the pelvic brim to the left psoas muscle, and ulceration of the left hypogastric vein with massive fatal hemorrhage; metastatic areas of granuloma inguinale in tibia, fibula, frontal bone and lungs; granuloma inguinale of pelvic lymph nodes; myeloid metaplasia of vertebral bone marrow and of spleen; phagocytosis of blood pigment in spleen and bone marrow; acute splenic tumor; cloudy swelling of liver and right kidney with small chronic focal necroses of the latter; necrosis of iliopsoas muscle; atrophy of heart, thyroid, adrenals; caseous tuberculosis of para-aortic, abdominal and tracheobronchial lymph nodes.

Unfortunately cultures from the vaginal tissue were unsatisfactory[1] and no blood was obtained prior to death for immunological studies.

DIAGNOSIS

There is nothing specific in the roentgenological findings leading to a diagnosis of granuloma inguinale. The lesions in the bones are essentially osteolytic with no reaction in the overlying periosteum, and with no regeneration of bone. Similar lesions are observed in the such osteolytic or osteoclastic diseases are metastatic carcinoma, leukemia, Hodgkin's disease, lymphosarcoma, multiple myeloma, and so forth, so that a diagnosis of granuloma inguinale of bones is impossible without clinical history and physical examination, assisted by laboratory studies. The location of the primary lesion, the persistent secondary anemia, and the marked con-

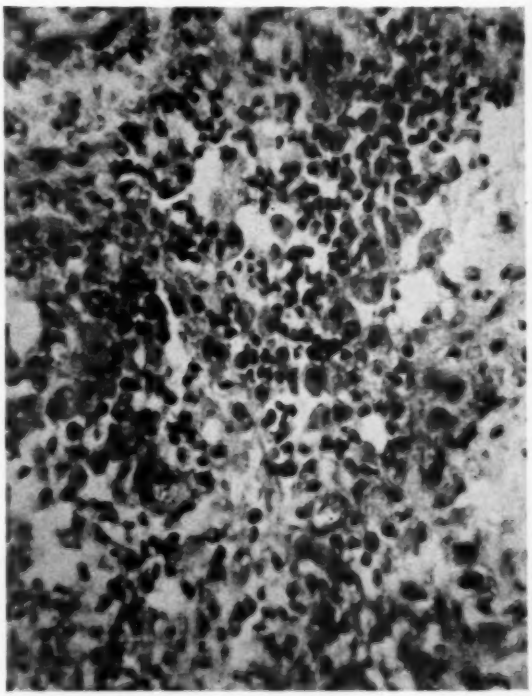

FIG. 8. Photomicrograph of lesion shown in Figures 6 and 7 to show the large macrophages and associated scattering of polymorphonuclear neutrophiles.

stitutional reaction with a continued hectic fever are noteworthy clinical features. Laboratory studies such as the complement fixation test, the precipitin test, and cutaneous reactions using antigens derived from cultures of the organism, *Donovania granulomatis*, on the yolks of chick embryo will probably be of distinct diagnostic value (Anderson, Goodpasture and De Monbreun[2]), but until these have been standardized and are readily available, the final diagnosis rests upon biopsy and the finding of the pathognomonic Donovan bodies and typical phagocytic cells in microscopic preparations.

SUMMARY

1. Disseminated granuloma inguinale of bones is an uncommon condition characterized roentgenologically by osteolytic areas in the long and short bones without visible reaction.

2. In none of the cases so far reported, including one of our own occurring in a female negro with a primary lesion in the cervix uteri, are the roentgenological changes specific for the diagnosis of this disease.

3. Osseous lesions of the type described in the presence of obscure genital lesions should suggest the possibility of disseminated granuloma inguinale.

4. Diagnosis of the disease is best established by biopsy with search for macrophages containing Donovan bodies, although cultures and immunologic studies promise to be of value.

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EOSINOPHILIC GRANULOMA OF BONE IN THE HAND

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EOSINOPHILIC granuloma has been reported in almost every bone except the bones of the hands or feet. The case to be reported in this paper is believed to be the first instance of this lesion in a small bone. Jaffe and Lichtenstein[1] in a very complete article on the subject as recently as 1944 state, "With the possible exception of the bones of the hands and feet, almost any bone apparently may be the site of a lesion in a case of eosinophilic granuloma whether the involvement of bone is single or multiple."

REPORT OF CASE

The patient was a white male, aged twenty-two, whose chief complaint was swelling of the distal phalanx of the third finger of the right hand. This had been present for two and one-half months, the last two weeks of which time there had been no significant progression of the swelling. He noticed that the swelling had occurred a few days following trauma by a baseball striking the end of his finger. There was no great amount of pain associated with the swelling. He presented himself to a dispensary where the lesion was treated for about one week as an infection (paronychia). Failure to improve, plus the fact that there were no indications of infection commensurate with the swelling, resulted in the dispensary sending the patient to hospital for roentgenological examination of the finger.

The roentgenogram showed a destructive lesion in the distal phalanx of the third finger involving the lateral two-thirds of the phalanx throughout practically its entire length. There was also a small radiolucent zone in the head and distal shaft of the third metacarpal. Another radiolucent area was noted in the distal radius about 5 cm. proximal to the radial joint surface. This area showed no expansion of cortex and no trabeculation. These lesions are seen in Figure 1, *A* and *B*.

Because the osteolytic lesion was multiple and because the general appearance of the roentgenograms suggested a possible cancerous growth, specifically multiple myeloma, or tumor metastatic to the skeleton, the skull, ribs and long bones and pelvis were roentgenographed. No other lesions were observed. The roentgenographic picture suggested some type of fibrocystic bone disease or possibly Schüller-Christian's disease. Because of the fact that there was rapid onset, meagerness of general manifestations clinically, multiple involvement of bones with only one lesion creating local symptoms and signs, the roentgenological report included the statement, "eosinophilic granuloma of bone cannot be ruled out."

The blood chemistry values were: Phosphorus, 2.7 mg. per 100 cc.; calcium, 11.3 mg. per 100 cc.; alkaline phosphatase, 6.5 Bodansky units.

The surgical intervention undertaken a few days later consisted of curettage of the distal phalanx of the third finger, for biopsy. The granulation tissue curetted from this lesion revealed histopathologically the picture of eosinophilic granuloma of bone as described in the following pathological report.

"*Gross Examination:* Gross specimen consists of multiple bits of solid grayish-white and grayish-brown tissue and several attached fragments of bone. The central areas are somewhat friable. Some marginal regions are more dense and section with increased resistance. Cystic spaces are not noted.

"*Microscopic Examination:* The lesion is very cellular and in most areas has a granulomatous appearance. Small blood vessels are numerous, and many capillaries are narrowed by swollen endothelial cells. There are several sites of old hemorrhage showing partial organization: occasional areas show accumulation of light brown pigment. Throughout the lesion there are scattered neutrocytes, plasma cells and lymphocytes; in some areas there are large numbers of eosinophiles, and a few of these cells can be found in all sections. The stroma in many places is composed of loosely arranged spindle cells, while at the margins of the sections there are regions of dense fibrous tissue. Other areas exhibit sheets of large cells having oval, somewhat vesicular nuclei and finely granular

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eosinophilic cytoplasm. A few scattered giant cells are seen in all parts of the lesion. These cells generally have 3–5 centrally placed nuclei, often overlapping or 'shelving' and the cell margins are indistinct.

"*Diagnosis and Comment:* This lesion is a benign but locally destructive process having many characteristics of a benign giant cell tumor. However, the x-ray, clinical and histopathological findings are not perfectly typical of this condition, and a diagnosis of eosinophilic granuloma of bone is suggested. (This is the opinion of the Fourth Medical Laboratory pathologist following an informal consultation.) C. F. Vorder Bruegge, Maj. M.C."

All the pathological material in this case was submitted to the Fourth Medical Laboratory whose report was as follows: "Concur with the diagnosis of eosinophilic granuloma of bone, Robert H. Quillinan, Capt. M.C., Chief of Pathology."

From this laboratory the pathologic material was forwarded to the Army Institute of Pathology, Washington, D. C., whose report was, "We agree with the diagnosis of eosinophilic granuloma of bone . . . although the lesion is not characteristic due to the great rarity of eosinophiles. The endothelial proliferation is very pronounced. J. E. Ash, Colonel, M. C., Director."

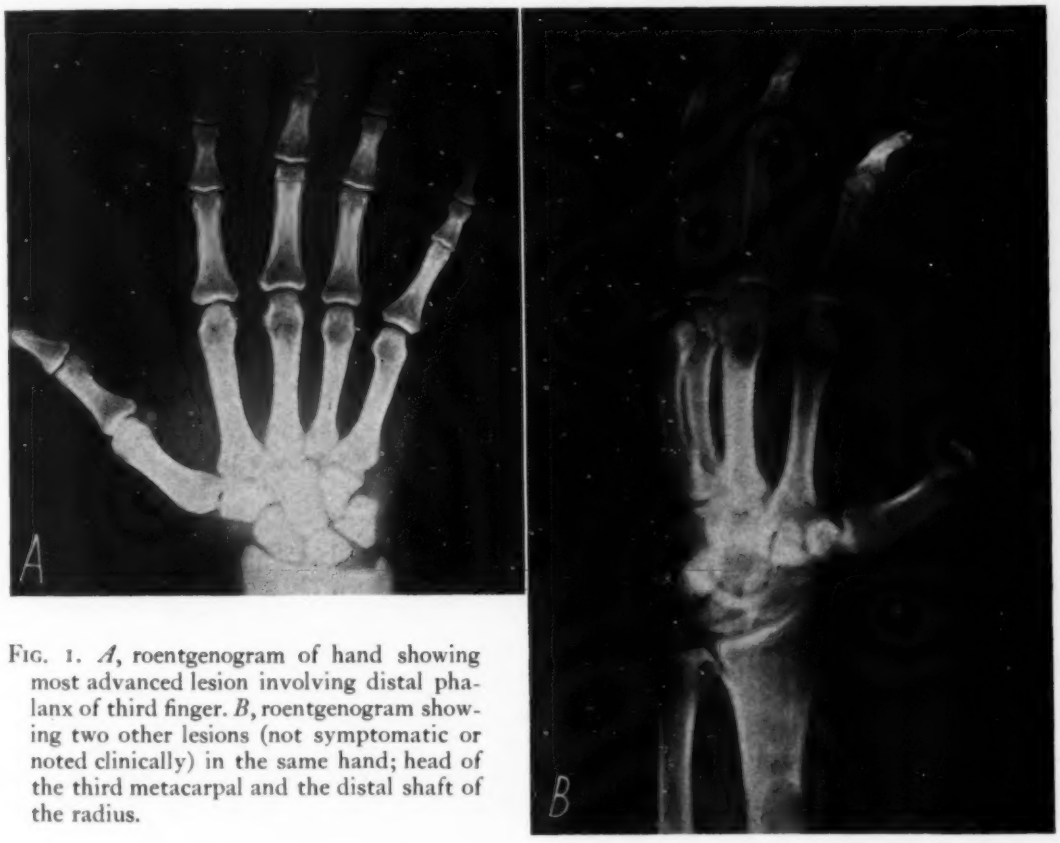

Fig. 1. *A*, roentgenogram of hand showing most advanced lesion involving distal phalanx of third finger. *B*, roentgenogram showing two other lesions (not symptomatic or noted clinically) in the same hand; head of the third metacarpal and the distal shaft of the radius.

It is interesting to note at this point that Green and Farber,[2] cited in the excellent review of eosinophilic granuloma by Jaffe and Lichtenstein[1] already referred to, have maintained that the lesion in passing from the acute to the older stage shows fewer eosinophiles until none may be found in the older lesion where an ingrowth of connective tissue predominates and large vacuolated mononuclear cells are seen.

Photomicrographs of the curetted material are shown in Figure 2, *A* and *B*.*

* Acknowledgment is made to the Army Medical Museum for the reproductions of the roentgenograms and photomicrographs in this case.

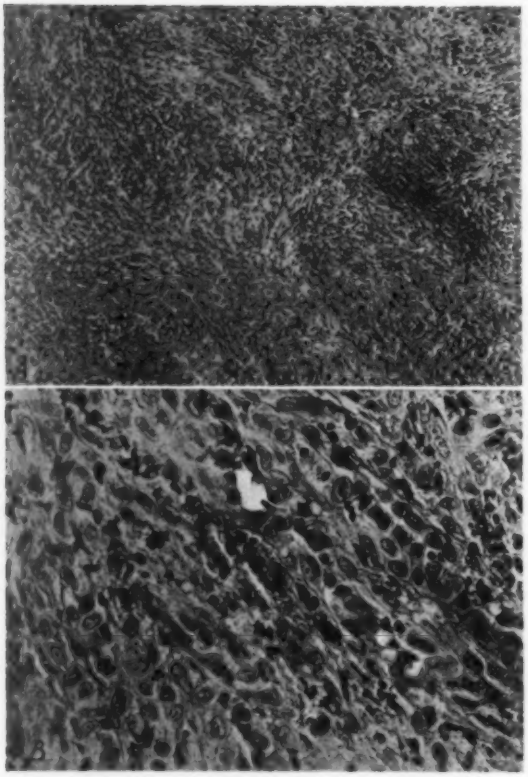

FIG. 2. *A*, photomicrograph (low power) of biopsy material from distal phalanx lesion shown in Figure 1*A*. The lesion is seen to be very cellular and to have a granulomatous appearance. *B*, photomicrograph (high power) of field in *A*. Note the presence of neutrocytes, plasma cells, lymphocytes, occasional eosinophiles and scattered giant cells with from three to five nuclei.

COMMENT AND CONCLUSIONS

Roentgenographically, this case showed lesions in three bones (distal phalanx of third finger, distal portion of third metacarpal and distal portion of the radius) of the right hand, although only one of them was giving rise to clinical symptoms when the patient was first admitted to the hospital. This makes it clear that even in cases in which there is supposedly only a solitary lesion, lesions may also be present in other bones, even though silent, and should be searched for roentgenographically.

Roentgenologically the individual lesion of bone presents itself as a small or large radiolucent area (Fig. 1, *A* and *B*). There is nothing characteristic or distinctive about the roentgenological picture of the individual lesion. The roentgen appearance suggests multiple myeloma, Ewing's tumor or tumor of bone metastatic to the skeleton, or, especially if lesions appear in the skull, it may strongly suggest Schüller-Christian's disease. Keeping in mind the fact that the disease occurs most frequently in children, adolescents and young adults; that it may be single or multiple; that if it is multiple there is usually one of the lesions much farther advanced and producing genuine clinical symptoms of a local nature only (systemic manifestations being entirely absent as a rule in cases with multiple bone involvement); that when there are symptoms referable to an affected bone, local tenderness and pain are the most common; and that if the part affected is rather superficial a local swelling without heat may be palpable, the roentgenologist can suggest the possibility of eosinophilic granuloma of bone from the roentgenogram.

The roentgenological diagnosis can only be suggested as a possibility since the definite diagnosis can only be made after histopathological study of the tissue obtained from the lesion. It is important, however, to suggest the possibility of this lesion for it is a benign one and solitary lesions which are curetted show gradual repair after this procedure alone. Green and Farber suggest the use of roentgen therapy to any remaining lesions in cases of multiple bone involvement. Spontaneous healing by resolution is also possible and has been proved by Jaffe and Lichtenstein.

This report demonstrates that the lesion can occur in the small bones of the hand and completes the list of portions of the skeleton known to be the site for a lesion of eosinophilic granuloma of bone.

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ROENTGENOGRAPHIC DEMONSTRATION OF SINUSES AND FISTULAE*

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THE use of radiopaque media to demonstrate cavities in the body dates back almost to the first use of the roentgen rays. Suspensions and pastes of heavy metal compounds were at first used to demonstrate depressions of the external surface, such as shallow cavities following wounds of the extremities. Since that time, the use of many types of contrast media has become a routine and integral part of roentgenological examinations. One of these substances is iodized oil, and its use in diagnostic procedures has reached into almost every branch of the medical and surgical specialties. Its use in the investigation of chronically draining sinuses and fistulae is a widely recognized procedure, but is often overlooked in specific cases in which it would give additional worthwhile information. Its use in certain fistulae, notably bronchial and biliary, has become a standard diagnostic procedure. It has been frequently used in the investigation of draining sinuses of the extremities following compound fractures incurred in the recent war, and has added pertinent information about the extent of abscess cavities, and the role played by foreign bodies or sequestra in perpetuating the drainage. The method has been thoroughly described and certain technical maneuvers outlined in the very excellent article by Gage and Williams.[1]

The use of iodized oil has been very helpful in the investigation of complicated war wounds seen on the Neurosurgical Section of an Army General Hospital that was treating a fairly large number of spinal cord and cauda equina injuries, in addition to an even larger number of major peripheral nerve injuries.

The method used was very simple. With aseptic technique a small rubber catheter or rubber-tipped syringe was gently inserted into the external opening and the iodized oil was injected under roentgenoscopic observation. After the full extent of flow was obtained, appropriate film studies were made. "Spot" roentgenograms taken during roentgenoscopy were found to be valuable as occasionally leakage of the oil may alter the findings before the remaining views have been obtained. Stereoscopic and lateral views were considered essential in the accurate localization of the extent of the sinus or fistulous tracts and their relations to neighboring structures. Occasionally the injection was combined with an air and barium study of the colon to more accurately localize the communication in instances of fecal fistulae. The injection of the iodized oil was not used in any cases suspected of having spinal fluid fistulae; there is usually little doubt as to diagnosis when such a fistula is present. This appeared to be one of the few contraindications to its use.

The cases presented here represent widely different conditions which have been demonstrated by the use of iodized oil injections. In many instances the diagnosis was in doubt and essential information was obtained. Only one patient had any ill effects and this consisted of a transient slight febrile reaction following the injection. In this instance, the external opening extended into a liver abscess which communicated with a small biliary duct.

CASE REPORTS

CASE 1. *Fecal fistula through the spine.* This twenty-seven year old American soldier was admitted to the Ashford General Hospital on October 11, 1944. On July 6, 1944, he sustained a

[1] Gage, H. C., and Williams, E. R. Radiological exploration of sinus tracts, fistulae and infected cavities. *Radiology*, 1943, *41*, 233–248.

* From the Radiological Service and the Neurosurgical Section, Ashford General Hospital, White Sulphur Springs, West Virginia.

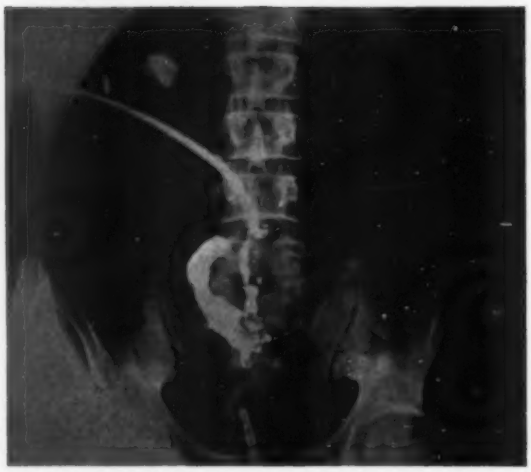

Fig. 1. Case 1. Injection of iodized oil into the draining sinus through the spine, showing the communication with a loop of intestine.

shell fragment wound of the spine in the region of the fifth lumbar vertebra. The next day a celiotomy was done and a perforation of the bladder and generalized peritonitis was found. Several days later he was found to have drainage of feces through the wound in the lumbar region; there was also drainage of feces through the suprapubic cystotomy tube. On July 29, 1944, an exploratory celiotomy was performed and a segment of ileum was found to communicate with the bladder. The source of drainage from the wound in the back was not found. Postoperatively the patient did well; however, the upper portion of the abdominal incision opened and drained feces.

Physical examination on admission to this hospital revealed a midline abdominal incision that was draining purulent material and feces through a small opening in its upper portion. There was a draining wound, 3 by 4 cm., in the region of the third lumbar spine. There was a partial paralysis of both lower extremities and anethesia over the distribution of all of the sacral nerve roots.

Fig. 2. Case 1. Lateral view showing the communication with a loop of large intestine and a sinus tract extending into the fifth lumbar interspace.

Fig. 3. Case 1. Retouched lateral view at time of second iodized oil injection.

On several occasions the patient had noted the passage of gas through the wound on the back. On February 5, 1945, the external opening on the back was injected with iodized oil. Roentgenograms revealed a sinus tract passing through the sacrum to enter the sigmoid colon; a second tract entered the fifth lumbar interspace (Fig. 1, 2 and 3).

On April 7, 1945, a transverse colostomy was performed; the drainage from the wound through the spine subsided. Complete healing of the fistula occurred within two months.

Case 11. *Urinary fistula through the spine.* This twenty-four year old American soldier was admitted to the Ashford General Hospital on October 3, 1944. On June 1, 1944, he sustained a shell fragment wound of the spine in the region of the fifth lumbar vertebra resulting in immediate paralysis of both lower extremities. Exploratory celiotomy on the day of injury revealed a large retroperitoneal hematoma, but no perforation of the peritoneal cavity. On July 20, 1944, an intravenous pyelogram revealed a shell fragment adjacent to the lower portion of the left ureter and there was some extravasation of the dye into the surrounding tissues (Fig. 4). On July 29, 1944, the shell fragment was removed and a retrograde catheter was passed into the ureter from the bladder.

Physical examination on admission to this hospital revealed several draining wounds on the back in the region of the fifth lumbar vertebra. The patient stated that one of these wounds would drain urine intermittently. There was a complete paralysis of the right lower ex-

Fig. 4. Case 11. Intravenous pyelogram on July 20, 1944, showing the foreign body at level of the lower left ureter with some extravasation of the dye.

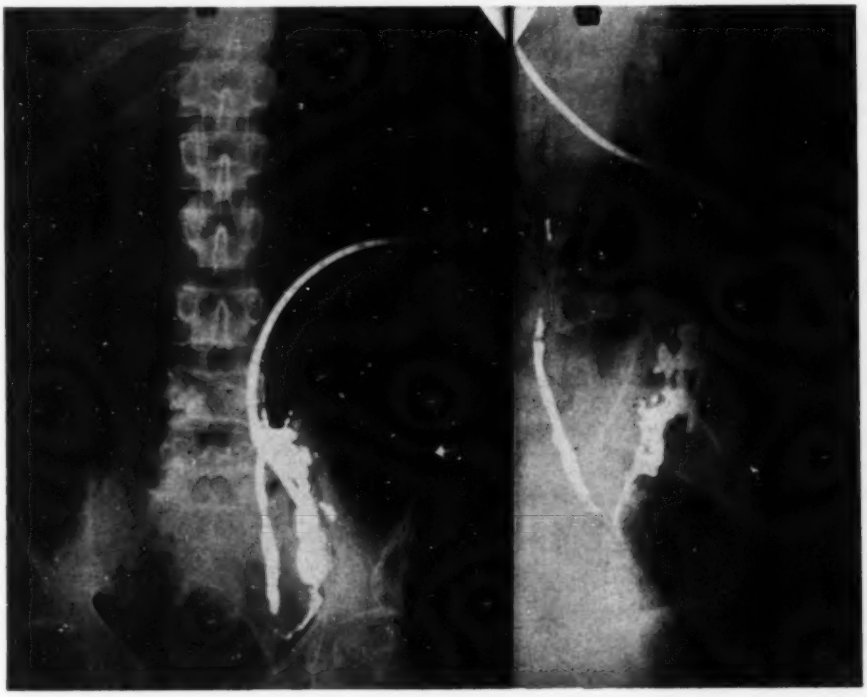

Fig. 5. Case 11. Anteroposterior and lateral views after injection of iodized oil showing the sinus through the sacroiliac joint communicating with the left ureter.

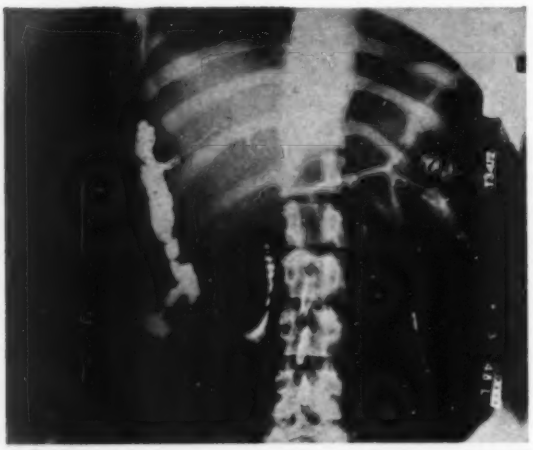

Fig. 6. Case III. Iodized oil injection of the draining sinus in the right upper abdomen showing an abscess cavity communicating with a small bile duct.

tremity and a partial paralysis of the left lower extremity.

A short time after admission to this hospital an intravenous pyelogram was done, but revealed no definite evidence of injury to the left ureter. Most of the wounds on the back eventually healed; however, one small wound continued to drain purulent material and, intermittently, urine. This was confirmed when methylene blue was given by mouth. On February 19, 1945, the external opening was injected with iodized oil. The oil passed through the region of the left sacroiliac joint, entered the left ureter near its distal end, and ascended the ureter to the level of the third lumbar vertebra (Fig. 5). On March 14, 1945, a left nephrectomy was performed and there was no further drainage of urine, although some purulent drainage from the wound on the back persisted. On May 2, 1945, the draining wound was explored and several sequestrated bone fragments were removed. Evidence of osteomyelitis in the left sacroiliac joint is present and some purulent drainage has persisted.

Case III. *Biliary fistula.* This twenty-two year old American soldier was admitted to the Ashford General Hospital on January 17, 1945. On October 17, 1944, he sustained a bullet wound of the abdomen and spine which resulted in immediate paralysis of the lower extremities. On October 18, 1944, celiotomy revealed a large laceration of the right lobe of the liver; this was packed and drained through a stab wound in the right upper quadrant of the abdomen. During the month before admission to this hospital, the patient had vomited after each meal, and had had considerable drainage of purulent material and bile from the opening on the abdominal wall.

Physical examination on admission to this hospital revealed an acutely ill patient with complete paralysis of both lower extremities. There was bile-stained purulent drainage from the wound in the right upper abdomen.

On February 5, 1945, the external opening in the right upper abdomen was injected with iodized oil. This revealed an abscess along the lateral portion of the right lobe of the liver (Fig. 6 and 7). Some of the oil entered a small bile duct and reached the common bile duct and duodenum.

On April 11, 1945, the abscess was opened in its lower portion and better drainage established. The patient's general condition improved, the drainage stopped and the wound was completely healed within two months.

Case IV. *Osteomyelitis of the spine; abscess in psoas muscle.* This twenty-six year old American soldier was admitted to the Ashford General Hospital on January 20, 1945. On September 14, 1944, he sustained a shell fragment

Fig. 7. Case III. Lateral view of the abscess cavity. Iodized oil entering the common bile duct and the duodenum.

wound of the spine at the level of the third lumbar vertebra resulting in partial paralysis of both lower extremities. On the day of the injury, a celiotomy revealed a perforation of the posterior portion of the ascending colon; exteriorization of this portion of the ascending colon was performed. Debridement of the wound on the back and laminectomy revealed that most of the roots of the cauda equina were intact. The laminectomy incision opened postoperatively and drained purulent material.

Physical examination on admission to this hospital revealed a laminectomy wound extending from the second lumbar to the fifth lumbar spines. There was an opening, 2 cm. in diameter, near the center of the wound which was draining purulent material. There was a paralysis of all of the nerve roots below the third lumbar segment. Roentgenograms of the lumbar spine revealed definite evidence of osteomyelitis in the bodies of the fourth and fifth lumbar vertebrae.

On April 11, 1945, the draining wound was injected with iodized oil. This demonstrated a communication with a large abscess cavity in the right psoas muscle (Fig. 8 and 9). On April 30, 1945, the laminectomy wound was explored

Fig. 8. Case iv. Iodized oil injection of the draining laminectomy wound. Osteomyelitis of the fourth and fifth lumbar vertebrae with a psoas abscess.

Fig. 9. Case iv. Lateral view showing the abscess cavity in the right psoas muscle.

and three small sequestrated bone fragments were removed. The drainage from the wound continued, and on August 14, 1945, the abscess in the right psoas muscle was drained through a paravertebral incision. The wound was healed at the end of three weeks. Drainage recurred six months later and will require further surgical treatment.

Case v. *Foreign body and draining sinus.* This twenty-six year old American soldier was admitted to the Ashford General Hospital on March 11, 1945. On November 2, 1944, he sustained a shell fragment wound of the spine in the region of the fifth lumbar vertebra, which resulted in immediate paralysis of the lower extremities. On November 3, 1944, laminectomy revealed that most of the roots of the cauda equina were intact and that only two roots on the right were actually severed. On November 6, 1944, the patient had evidence of meningitis and was given penicillin intrathecally. He continued to have an elevated temperature for the next two weeks and developed a moderate amount of purulent drainage from the laminectomy wound. There had been progressive improvement of the motion in the lower extremities since the time of the injury; however, the

FIG. 10. Case V. Iodized oil injection of the draining laminectomy wound. Collection of oil in the neural canal.

drainage from the laminectomy wound persisted.

Physical examination on admission to this hospital revealed a laminectomy wound extending from the fourth lumbar to the second sacral spines. There was a small area about 0.5 cm. in diameter near the center of the wound which was still draining purulent material. The patient was able to accomplish all movements of the lower extremities weakly. On April 20, 1945, the draining sinus was injected with iodized oil. This revealed a small abscess cavity in the region of the neural canal (Fig. 10).

On April 30, 1945, the sinus tract was explored and a piece of soft rubber drain, 5 inches long, was found in the region of the neural canal. Healing was complete within three weeks.

CASE VI. *Foreign body and draining sinus.* This twenty-five year old American soldier was admitted to the Ashford General Hospital on February 12, 1945. On September 17, 1944, he sustained a machine gun bullet wound of the right buttock, which resulted in partial paralysis of the right lower extremity. On September

FIG. 12. Case VI. Iodized oil injection of the draining sinus showing a tract through the sciatic notch into the region of the foreign body.

FIG. 11. Case VI. Large foreign body in pelvis. Draining sinus in the right buttock.

18, 1944, a celiotomy revealed a large retroperitoneal hematoma, but there was no perforation of the peritoneal cavity. The bullet was not removed. Several days later there was some purulent drainage from the wound in the right buttock. The drainage from the wound persisted.

Physical examination on admission to this hospital revealed a scarred wound on the right buttock, 20 cm. in length. There was a moderate amount of purulent drainage from a small opening near the center of this wound. There was a partial paralysis of the right lower extremity. Roentgenograms revealed a fracture of the inferior margin of the ilium near the right sacroiliac joint and a metallic foreign body (bullet), 4 by 1 cm., lying immediately inferior

and 4 cm. anterior to the third sacral foramen on the right (Fig. 11).

On April 5, 1945, the draining sinus was injected with iodized oil and revealed a tract to extend inferior and anterior to the right sacroiliac joint and into the region of the bullet (Fig. 12).

On May 19, 1945, an air contrast barium enema demonstrated that the foreign body was posterior and slightly to the right of the sigmoid colon. On June 12, 1945, the bullet was removed transperitoneally. The drainage from the sinus tract in the right buttock diminished. Healing was complete within three months.

SUMMARY

The use of iodized oil for the investigation of chronically draining sinuses and fistulae has been discussed.

Six cases have been presented in which essential information was obtained by this method of examination.

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VOLVULUS OF THE STOMACH COMPLICATED BY PEPTIC ULCER

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VERY little has been written concerning volvulus of the stomach, and many authors have dismissed this subject with a simple notation here and there. It is intended in this article to describe the condition more clearly, and present an interesting case in which some of the salient diagnostic features are demonstrable. By the term volvulus, we infer a pathological twist, bend, torsion or rotation of an organ on its axis. This condition has been ascribed to several factors and is usually concomitant with distention and displacement of the colon, especially in relationship with interposition of the large intestine, where the colon ascends upward anterior to the stomach, and where, in this upward ascent, it travels between the anterior surface of the liver and the diaphragm, otherwise known as anterior hepatodiaphragmatic interposition. In this continued upsweep, the greater curvature of the stomach is drawn proximally in front of the lesser curvature, by the transverse mesocolon and, as a result, lies at a higher level than the lesser curvature, almost on a plane with the cardiac end of the stomach. The pylorus and lesser curvature, depending on the axis of rotation, may not be altered to any great extent as to their position and at times may be more or less fixed, the ligament of Treitz possibly acting as a restraining influence in the rotation process. This situation is referred to as a volvulus of the stomach on its cardiopyloric axis. In this position, the normal longitudinal rugae of the stomach no longer lie in its long axis, but instead appear obliquely or transversely across the stomach, due to the rotation of this organ. The peristaltic waves are also altered, in that the cycle at first presents somewhat deeper and more vigorous contractions in attempting to propel the food, but as the condition becomes chronic these waves become more shallow, and soon there develop periods of quiescence which ultimately lead to atony, delayed functional activity, and increased food retention. With rotation of the stomach, the blood supply of this organ, namely the gastric and pyloric arteries which supply the lesser curvature, and the right and left gastroepiploic arteries which supply the greater curvature, is also disturbed, some constriction of these vessels taking place, with subsequent ischemic changes. The lymphatic channels and nerve structures are commensurately involved and there may be changes in their course, with possibly some backflow of the lymph associated with subjective manifestations. These variations in position, blood, lymph and nerve supply are definitely predisposing causes for superimposed pathological processes such as ulceration, obstruction, pyloric stenosis, and so forth. The case illustrated in this article shows evidence of secondary changes consisting of pyloric stenosis accompanied by a duodenal ulcer, which was confirmed by operation.

Other causes of volvulus of the stomach may be associated with intra- or extragastric pathologic conditions, which may be either congenital or acquired. Among these causes are listed congenital anomalies, diaphragmatic herniation, eventration, cancer of the cardia, benign gastric tumor, hour-glass stomach, phrenectomy, and so forth. Thus it can be seen that whenever there is a change in the status quo of the stomach and surrounding structures, whether it is due to changes in negative pressure, position, alteration due to infiltration, or loss of normal organal balance, or a weakening of the surrounding structures about the stomach, there is always the possibility of the occurrence of a volvulus. Sometimes the hepatic flexure and proximal transverse colon may pass up behind

the liver, distorting the stomach. At other times, the distal transverse colon may be so rotated that it ultimately comes to lie to the right of the fundus of the stomach, crossing in front of it to form a transverse arc for the splenic flexure, this latter portion pressing against the diaphragm, and then proceeding distally to form the descending colon.

There are several types of volvulus of the stomach, depending on the type of rotation and its duration. These may be (1) acute, due to sudden torsion of the fundus of the stomach, often associated with esophageal obstruction, (2) chronic, as the name implies, of longer duration, the etiological basis of which has been heretofore described (3) cardiopyloric, also herein discussed, or (4) transverse. Of these types, only the transverse has as yet not been explained. In this variety, the stomach bends on itself in the form of a "U," or on a coronal axis, with the antral region of the stomach swinging upward, in front of the pars media, to reach the left diaphragm near the cardiac portion of the stomach, so that the antrum and fundus are almost in apposition. In this instance, the pylorus may point to the left of the spine and slightly downward. On close inspection, two barium fluid levels with corresponding air pockets are seen, one in the antral region, and the other in the fundus of the stomach. This type of volvulus is possible only if the duodenum is elongated, or if it has lax attachments, or if an actual mesentery is present.

Other types of volvulus of the stomach include (*a*) the partial type, where the greater curvature of the stomach rotates incompletely, with the anterior face of the stomach displaced partially upward and in this case it is the pyloric portion which rotates, while the cardiac end of the stomach remains in situ, and (*b*) the complete type which consists of a twisting of the entire stomach around the cardiopyloric axis, so that the greater curvature now lies under the diaphragm and the posterior wall of the stomach faces forward.

Volvulus of the stomach has also been classified as either of the mesenteric axial type, organic axial type, or mixed type. These varieties have been included in this paper under cardiopyloric axial and transverse axial types or a combination of both.

In all cases of volvulus, whether the rotation is on the transverse or longitudinal axis, there is an apparent stretching of the stomach, causing an increase in its total length. This seems to be due to the constant stress and strain placed upon the stomach, and, in order to accommodate itself to its new position in relation to the surrounding structures, it elongates in the direction of the axial volvulus. When there is a laxity of the ligamentous structures, or the presence of a visceroptosis, or an overdistended stomach, or short omental attachment, it is then that a predisposition to volvulus occurs.

The chief roentgenological signs of volvulus of the stomach depend more on the type of axial rotation. If a volvulus occurs on the transverse axis, then we must look for (*a*) the double fluid level, capped by gas, in the antral and cardiac regions, with the antrum lying in front of and reaching almost on a plane with the fundus of the stomach, (*b*) the path of the rugae, which can be traced from cardia to antrum, assuming an oblique "U," with the maximum bend within the pars media, (*c*) a more mobile and ectopic position of the stomach, (*d*) an elongated esophagus, (*e*) sometimes a cascade appearing stomach, and (*f*) resultant secondary obstructive and stenotic signs.

If the stomach is rotated on its long or cardiopyloric axis, then the roentgenological signs to be sought for would be (*a*) an elongated stomach rotated on its cardiopyloric axis, (*b*) an elongated esophagus, (*c*) an anterior surface of the stomach facing posteriorly, while the posterior surface faces anteriorly, (*d*) a greater curvature of the stomach which lies almost against the diaphragm or to the right and front of the fundus and lesser curvature, (*e*) longitudinal rugae of the stomach becoming

FIG. 1. The barium-filled stomach is somewhat distended, the pyrlorus tit-like, and no barium is seen in the duodenum or elsewhere in the gastrointestinal tract.

transverse in direction, especially in the pars cardia and media, (*f*) a twisted stomach with the maximum narrowing within the vicinity of the twist, (*g*) a pylorus and duodenum located to the left of the midline, facing the pars cardia and directed either upward or downward, (*h*) an existing interposition of the stomach between the left lobe of the liver and diaphragm, (*i*) an associated obstructive or stenotic sign, and (*j*) a concomitant associated pathological condition, such as ulceration of the stomach, duodenum and esophagus.

The following case is presented, which portrays a volvulus of the stomach with rotation on its cardiopyloric axis associated with a secondary peptic ulcer.

REPORT OF CASE

A. M., male, aged sixty, complained of gastric distress which had been growing progressively worse for the past two years. Prior to that time, he had occasional symptoms of distention and discomfort which were of an evanescent character. The pains grew worse, more so at night, and occurred with regularity about one and one-half hours after meal time and would be relieved by either taking milk or alkalis, or by self-induced emesis, the latter always containing food of meals partaken a day or two before. The pains were often so severe at night that it would wake him from his sleep. There had been a 30 pound weight loss in the past year, and the patient experienced a sense of weakness, plus a phobia of food intake, since it was always followed by emesis. He had a sallow complexion and appeared cachectic. Gastric analysis revealed evidence of a hyperacidity with no free blood.

Administration of barium by mouth revealed the contrast medium entering an elongated esophagus, which appeared to enter the cardiac end of the stomach, more posteriorly and farther to the left than normally, and which did not seem to press against the greater curvature. The barium fell to the bottom of the stomach, due to marked hypersecretion. The barium proceeded to the pylorus, where the pyloric canal appeared tit-like in character, and where the

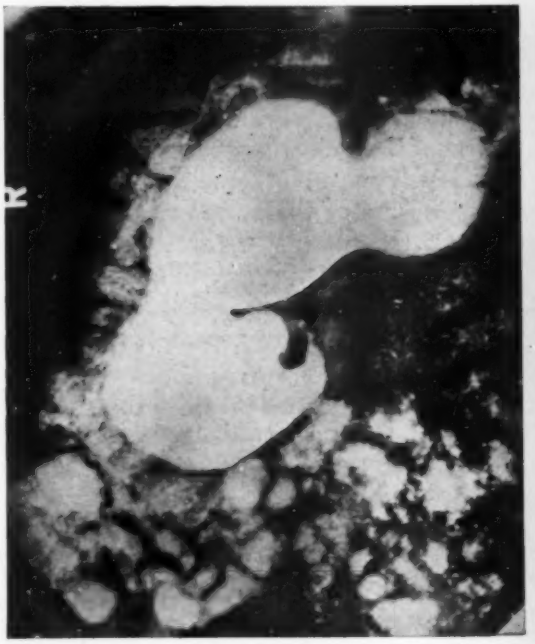

FIG. 2. The rugae in the pars cardia have a transverse direction. The tit-like pylorus is more prominent at this time. The position of the pylorus and duodenum is to the left of the midline and the stomach.

contrast medium was held up for about twenty minutes before it was seen to leave the pyloric ring. The stomach appeared somewhat distended, more so than is usually seen when an excessive amount of barium is given (Fig. 1).

The peristaltic waves seemed to be forceful at first but soon lost their vigor and became somewhat sluggish. The course of the rugae were definitely altered, assuming a transverse pattern in the upper third of the stomach, spiralling around the body and again becoming more horizontal with a slight upward trend as the pylorus was approached. The antrum, pylorus and duodenum lay to the left of the pars media. In spite of the general appearance of the stomach, the mobility of this organ appeared fair except for the pyloric and duodenal area (Fig. 2).

Forceful manipulation of the pylorus failed to immediately express any of the barium contents into the duodenum, but after a time, a small amount of the contrast medium was seen to enter the duodenal bulb. The pyloric ring itself was eccentric in position. At no time during the examination was a well filled cap visualized, but a constant fleck was seen near the base of the bulb, which was persistent and which represented the true ulcer niche. On roentgenoscopy definite pain, tenderness and fixation were elicited over this area. The convergence or radiance of the mucosal folds or so-called halo could not be demonstrated. On the roentgenograms the niche is clearly delineated (Fig. 3).

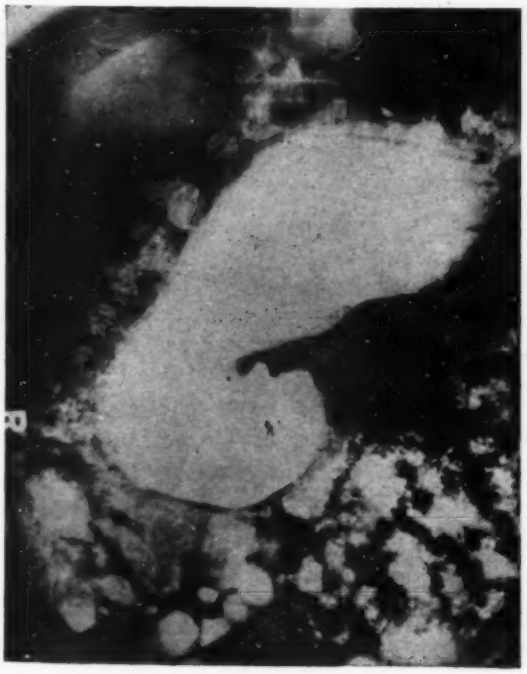

FIG. 3. The pinhead-sized barium-filled ulcer crater is seen which was constant throughout the entire examination.

FIG. 4. A large six hour gastric retention is seen. The course and position of the colon are outlined by arrows.

At the end of six hours, a large gastric residue was seen, with the head of the barium column advancing toward the splenic flexure, and the tail of the contrast meal in the terminal loops of the ileum. The distal colon was outlined with gas. It is interesting to note the course of the colon as shown in Figure 4. The hepatic flexure assumes the normal position, but the proximal half of the transverse colon forms a sharp bend near the midline and the colon traverses an upward path lying in front of and to the right of the rotated stomach, or greater curvature. It proceeds obliquely upward and across in front of the fundus of the stomach, finally making another bend to form the splenic flexure and thence downward toward the sigmoid and rectum (Fig. 4).

To demonstrate the position of the rotated stomach, a radiopaque Miller-Abbott tube was introduced and the pathway of the contrast

FIG. 5. The radiopaque Miller-Abbott tube definitely outlines the stomach pathway, indicating the presence of vovulus on its cardiopyloric axis.

medium could be readily followed by noting the course of this tube. The metal tip is seen to rest against the pyloric ring. In Figure 5, the entire colon is outlined by its gaseous content.

The patient was subsequently operated on, and a volvulus of the stomach on its cardiopyloric axis and a duodenal ulcer were found. A subtotal gastrectomy was performed, with the patient making a very good recovery.

CONCLUSION

1. Volvulus of the stomach is an uncommon condition, but when it does occur, its rotation is chiefly either in the transverse or longitudinal cardiopyloric axis.

2. The salient diagnostic features of this condition are described.

3. A case of volvulus of the stomach on its cardiopyloric axis associated with a duodenal ulcer is presented, the diagnosis of which was confirmed by operation.

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ROENTGENOLOGICAL ASPECTS OF BATTLE INJURIES OF THE CHEST

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BATTLE injuries of the thorax are of frequent occurrence. It is estimated that they are responsible for about one-quarter of the battlefield deaths. This high immediate mortality rate results in a comparatively low base hospital incidence where a little more than 1 out of 12 cases are represented by chest wounds.[2] However, the most significant feature, the quality or type of thoracic wounds presented by each individual case, cannot be adequately reflected in relative statistics. Thanks to the remarkable advances in medicine, surgery, anesthesia, and medical evacuation, we are encountering today a variety and severity of intrathoracic injuries that rarely survived in past decades.

The treatment of thoracic wounds in the field is devoted primarily to life-saving measures. Of importance very often equal to the control of shock, hemorrhage, and sepsis is the correction of cardiorespiratory imbalance as occasioned by open and tension pneumothorax, cardiac tamponade, stove-in chest, massive hemothorax or massive atelectasis. For the accurate diagnosis and proper treatment of these conditions, as well as for the demonstration of foreign bodies and thoraco-abdominal wounds, the surgeon depends a great deal on roentgen findings.

It is at the base hospital, where definitive treatment is carried out, that roentgenography assumes a wider, if less dramatic, scope in the management of thoracic injuries. Here, not only are retained foreign bodies localized and fractures of the thorax described, but such attending conditions of thoracic wounds as pneumothorax, hemothorax, pulmonary contusion, empyema, and so forth, are carefully evaluated.

The following techniques (Table 1) are

TABLE I

TECHNICAL FACTORS UTILIZED IN STUDY OF THORACIC INJURIES IN BATTLE CASUALTIES
Power: General Electric 200 milliampere, fully rectified unit, double focus tube
Screens: Eastman fine grain
Film: Eastman blue binder

	Postero-anterior Chest Erect; Routine Technique	Lateral Chest, Erect Routine Technique	Oblique Views Erect	Recumbent Lateral Antero-posterior Projection	Recumbent Antero-posterior	Recumbent Lateral Chest	Overpenetrated Films			
							Postero-anterior Erect	Lateral Erect Wafer Grid	Potter-Bucky Grid Antero-posterior Recumbent	Potter-Bucky Grid Lateral Recumbent
Anode-screen distance	72"	72"	72"	36"	36"	36"	72"	72"	36"	36"
Milliamperes	150 (large focus)	150	150	150	150	150	150	150	30 Small focus	30 Small focus
Milliampere-seconds	15	15	15	15	10	15	15	50	120	90
Time	1/10 sec.	1/10 sec.	1/10 sec.	1/10 sec.	1/20 sec.	1/10 sec.	1/10 sec.	1/3 sec.	4	3
Average kilovolts (peak) (depending on width of part)	68	85	78	62	64	70	78	85	66	80

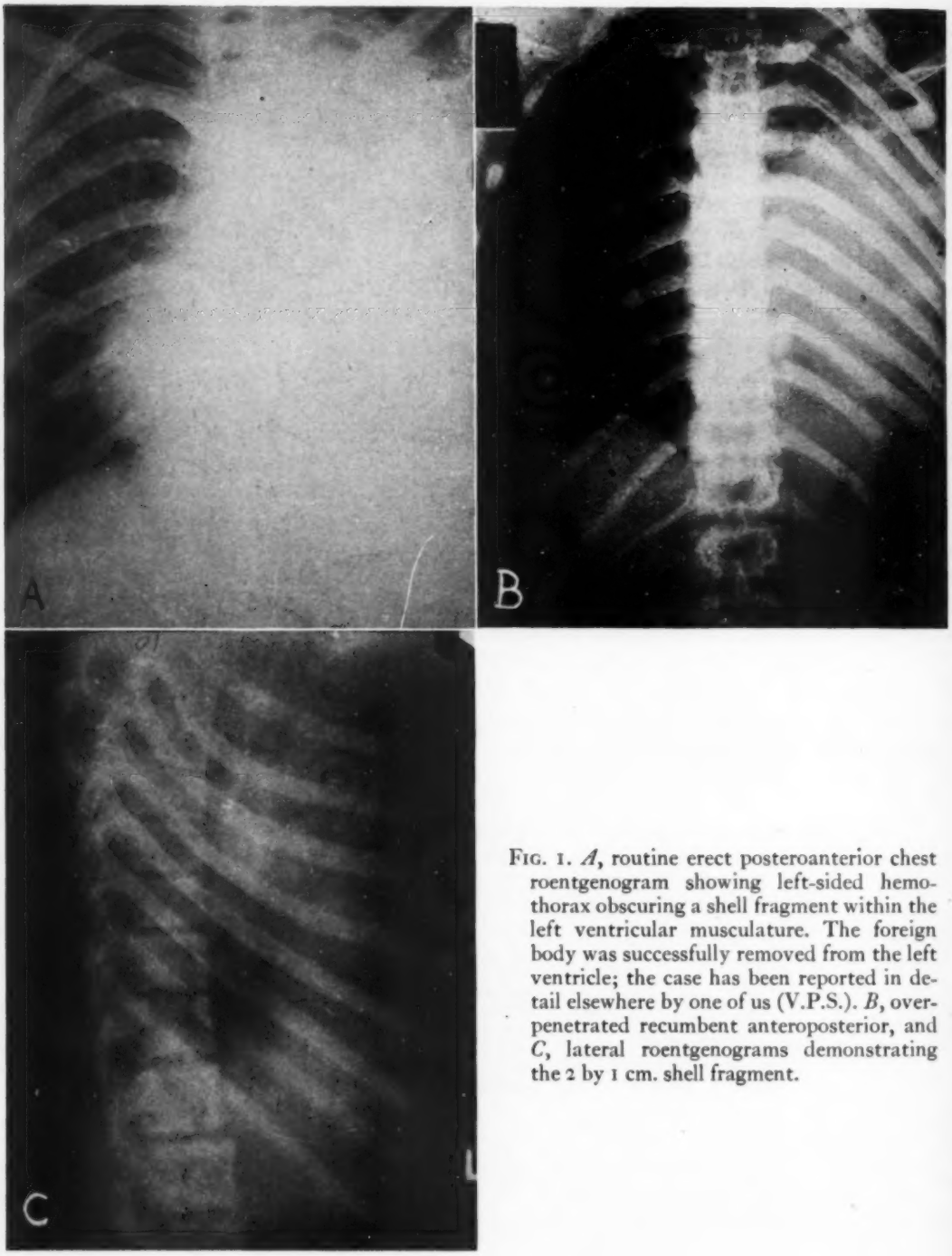

FIG. 1. *A*, routine erect posteroanterior chest roentgenogram showing left-sided hemothorax obscuring a shell fragment within the left ventricular musculature. The foreign body was successfully removed from the left ventricle; the case has been reported in detail elsewhere by one of us (V.P.S.). *B*, overpenetrated recumbent anteroposterior, and *C*, lateral roentgenograms demonstrating the 2 by 1 cm. shell fragment.

available for the study of intrathoracic pathology:

1. Routine upright posteroanterior and lateral views
2. Overpenetrated films
3. Anteroposterior views in the lateral recumbent position
4. Right and left oblique views
5. Stereoscopy
6. Roentgenoscopy

In every instance possible, regular upright roentgenograms should be taken so that free fluid can gravitate to the bases, otherwise, in the prone position, a small amount of fluid can obscure an entire hemithorax and give the impression of massive involvement. In those instances in which it is not feasible to have the patient sit up, the films should be marked "recumbent." Overpenetrated films are frequently indicated to identify foreign bodies by contrast against massive pleural effusion (Fig. 1). Anteroposterior views in the lateral recumbent position are useful in visualizing fluid levels in loculated or encapsulated collections (Fig. 8, *C* and *D*) while oblique and lateral views are important in localizing accumulations in relationship to pleural reflections (Fig. 8*B*).

Roentgenoscopy is of inestimable value in the study of foreign bodies, fluid levels and lung re-expansion, in the observance of diaphragmatic mobility and costal cage motion, in detecting areas of pleural exudate and sites of adhesions, and in the study of cardiac configuration and activity. Stereoscopy has proved a most helpful adjunct in the precise localization of foreign bodies.

This report is based on approximately 200 cases of thoracic injuries observed at this base hospital. By means of representative cases the roentgenological aspects of the conditions encountered are emphasized.

Foreign Bodies. Foreign bodies traumatizing the chest consist of bullets, shell fragments, projectiles, and missiles of various types. Due to their irregular shape and jagged edges, shell fragments are more readily retained within the tissues and are more prone to bring in with them particles of clothing and foreign material than do the high velocity bullets. They therefore produce a greater extent of trauma and a higher morbidity and mortality rate.

Metallic particles may strike the thorax directly or tangentially; their course is often biazarre, depending on the mass, size, direction and velocity of the foreign body and the structures met with. In one case a 2 by 1 cm. shell fragment entering the right infraclavicular area was recovered from the right deltoid muscle just below the acromion process. In many instances foreign bodies entering the upper thorax were found situated at or below the diaphragm on the same or the side opposite to the point of entry. Cases of unusual travel are legion.

The determination of the exact size and location of these fragments are of extreme importance since upon this information may depend the entire question of surgical exploration or the surgical approach to be employed. Posteroanterior and true lateral views, routine or overpenetrated, are ordinarily sufficient to demonstrate and localize foreign bodies anatomically (Fig. 1, 9 and 12). Depth measurements have not proved of practical value in this regard. Kymography is useful as an adjunct in those instances where intracardiac foreign bodies are suspected.

Vital information concerning foreign bodies can be gathered from roentgenoscopy. A fragment which moves with the lung markings is probably intrapulmonary; by consideration of the normal pulmonary landmarks, it can usually be localized to a given lobe. A fragment lying in proximity to the heart or great vessels will have an indirect motion transmitted from these structures; when impacted within the cardiac muscle (Fig. 1) it will obtain a characteristic to and fro motion synchronous with the heart beat and independent of respiratory excursion. A fragment lying free in a heart chamber will display a dancing type of motion and will move with change of position of the patient.

Finally, steroscopy proves helpful in localizing foreign bodies in intimate relationship to the thoracic cage. Figure 2 shows a large crescent-shaped metallic fragment reported, in the initial roentgenograms, to be in the left infraclavicular region. The wound of entrance was at the lateral border of the lower third of the left scapula. Stereoscopy demonstrated that the fragment was lying transversely across the axillary surface of the left second and

FIG. 2. Extrathoracic shell fragment measuring 4 by 1 cm. and comminuted fractures of the second to fifth left ribs.

third ribs. This localization facilitated the extrication of the foreign body, which was found, moreover, to be infringing on the axillary artery.

Injury to the Bony Thorax. Damage to the bony thorax is a common occurrence in battle casualties. This may vary from simple fracture to severe comminution of bone (Fig. 3). The ribs, scapula, clavicle, sternum, and the dorsal spine are all prone to injury. Pulmonary or pleural trauma is generally associated.

Pulmonary Contusion (Parenchymal Hematomas). Injury to the pulmonary parenchyma is invariably manifested by localized or diffuse areas or interstitial or intra-alveolar hemorrhage. The injury sustained by the lung may be in direct proportion, or bear little relationship to the force of trauma.

Roentgenologically, pulmonary contusion is represented by one or more areas of opacity of bizarre distribution and irregular outline (Fig. 3 and 4). A mild degree of atelectasis may be associated.

Differential diagnosis includes blast injury and pneumonia. With respect to the former, pulmonary contusion is almost always unilateral and is associated with direct trauma. Pulmonary contusion, furthermore, resolves more slowly than either blast injury or pneumonia.

Figure 4 illustrates the average rate of resolution in a case of pulmonary contusion.

This soldier, aged twenty-five, sustained an unusual tangential gunshot wound of the chest on April 18, 1945. The bullet entered the soft tissues over the right scapular region, traversed the entire back, inflicted a comminuted fracture of the left scapula with left hemothorax and parenchymal hematomas, and made its exit through the left arm without fracturing the humerus or injuring vessels or nerves. Thoracentesis on April 18 procured 400 cc. of blood; an additional 400 cc. of blood was aspirated on April 22. Convalescence was thereafter uneventful. The hematomas resolved in one month.

Subcutaneous Emphysema. Disruption of the pleurae in association with laceration of the parietes due either to perforating wounds or fractured ribs frequently results in subcutaneous emphysema. Extensive involvement often occurs in conjunction with a tension pneumothorax which continues to force air into the fascial planes of the chest wall until the tension is relieved, spontaneously or by surgical intervention.

Roentgenologically, subcutaneous em-

FIG. 3. Severely comminuted fracture of right clavicle and scapula; fracture-dislocation of the shoulder joint. The opacity overlying the right upper pulmonary field is attributed to pulmonary hematomas.

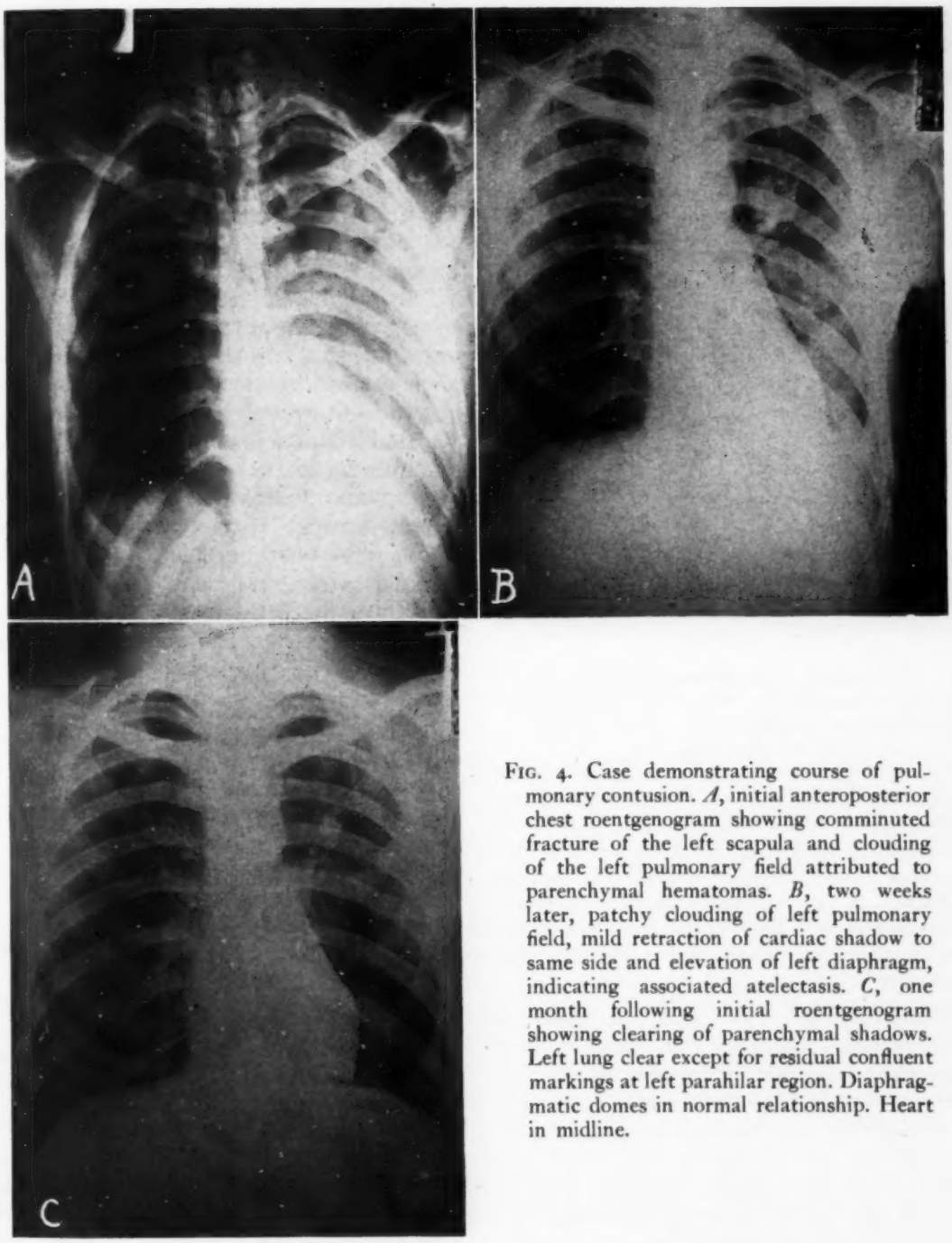

Fig. 4. Case demonstrating course of pulmonary contusion. *A*, initial anteroposterior chest roentgenogram showing comminuted fracture of the left scapula and clouding of the left pulmonary field attributed to parenchymal hematomas. *B*, two weeks later, patchy clouding of left pulmonary field, mild retraction of cardiac shadow to same side and elevation of left diaphragm, indicating associated atelectasis. *C*, one month following initial roentgenogram showing clearing of parenchymal shadows. Left lung clear except for residual confluent markings at left parahilar region. Diaphragmatic domes in normal relationship. Heart in midline.

physema appears as streak-like areas of increased radiolucency. It may be necessary to differentiate by clinical and laboratory means localized traumatic subcutaneous emphysema from the emphysema caused by anaerobic infection.

Figure 5 demonstrates a case of subcutaneous emphysema in a nineteen year old soldier who was wounded in action August 15, 1944, sustaining a sucking gunshot wound of the chest. The bullet entered the twelfth left rib in the posterior scapular line. In the evacuation hos-

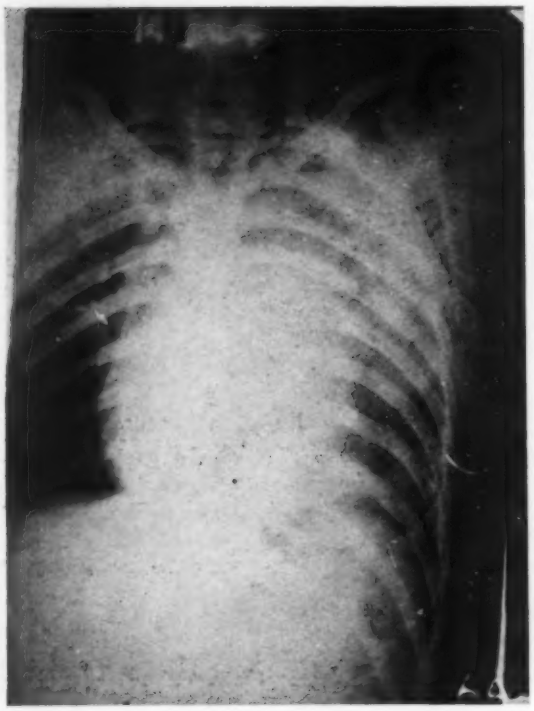

FIG. 5. Subcutaneous emphysema involving the left supraclavicular region and lateral chest wall. Note moderate left hemothorax.

pital, tight chest closure was effected, 800 cc. of blood was aspirated from the left chest, and water-seal drainage was instituted and maintained for two weeks, until all signs of a tension pneumothorax subsided. Subcutaneous emphysema was in evidence from the cervical region down to and including the scrotum. Two weeks were required for the complete resorption of the emphysema.

Pneumothorax. Practically all cases of penetrating wounds of the chest suffer a pneumothorax, air being admitted from the exterior. Pneumothorax may also occur in non-penetrating wounds of the chest. In these instances, air reaches the intrapleural space from the alveoli through visceral pleura and pulmonary parenchyma lacerated by a fractured rib. In either mechanism, if the wounds promptly seal off, minimal pneumothorax, unless looked for early, may not be discernible due to early resorption of air.

Pneumothoraces display characteristic roentgen features: a brilliantly illuminated hemithorax devoid of lung markings, obscured medially by the collapsed lung, and producing a mild displacement of the cardiac shadow to the opposite side. The extent of displacement and diaphragmatic depression serve to indicate the amount of pressure under which the air is encased in the thorax. However, manometric readings are required for accurate measurement of a pneumothorax under tension.

Tension pneumothorax results when the tissues through which the air is admitted act as a one-way valve, permitting the ingress of air into the thorax with each inspiration, but prohibiting egress with expiration. Although both open and tension pneumothorax are adequately cared for in the advanced echelons, occasionally the operative causes may recur. A fragmented rib, in transportation, may puncture the visceral pleura; infection may re-open a wound, or pressure changes relative to air evacuation may be causative.[6] The following is a case of tension pneumothorax:

This soldier, wounded February 26, 1945, sustained multiple shell fragment wounds. One fragment imposed a tangential wound of the chest, shattering the right scapula, and fracturing the third and fourth right ribs. There was no penetration of the thorax by shell fragments. Hemothorax was present. The wounds were debrided and intercostal drainage instituted. Blood, plasma, oxygen, sulfadiazine and penicillin were administered. On the fourth postoperative day the intercostal tube was removed and the patient was evacuated to another field installation where, on the following day, he experienced marked dyspnea. A roentgenogram revealed the presence of a pneumothorax; 750 cc. of air was aspirated under tension. The following day a small catheter was inserted in the third right anterior interspace, and the patient was air-evacuated to this base hospital. On admission, he was cyanotic and markedly dyspneic. A roentgenogram showed the reappearance of the tension pneumothorax (Fig. 6) which produced an intrapleural pressure of plus 6 cm. of water. Water-seal drainage was instituted. The following day roentgen examination showed almost complete expansion of the affected lung with an intrapleural pressure reading of minus 6 cm. of water.

Hemothorax is a natural sequel to perforating chest wounds, and is invariably found as a hemopneumothorax. It also occurs with non-penetrating wounds, including blast injury and pulmonary contusion, and altogether appears in approximately 75 per cent of battle casualties involving the chest. The bleeding may come from a lacerated intercostal or internal mammary vessel or, commonly, from the lung parenchyma. Patients with hemorrhage from the hilar or large intrathoracic vessels rarely survive. The exertion of positive pressure by the accumulating intrathoracic blood and the relatively low pressure of the pulmonary circulation are factors which serve to arrest bleeding, particularly from the lung parenchyma. However, hemorrhage may recur, due to a rise in blood pressure, movement of splintered ribs during transportation, or too early aspiration of a hemothorax before complete sealing off of the injured parenchyma has taken effect. Moreover, because blood serves to irritate the pleurae, recurring pleural effusions are not at all infrequent, necessitating aspirations as late as two to three weeks following the initial evacuation of blood. It is therefore imperative to observe these cases closely, both clinically and by follow-up roentgen study.

Roentgenologically, the diagnosis of hemothorax is presumptive since free blood, pus and serum cannot be differentiated on the roentgenogram. The roentgenologic interpretation, therefore, will be concerned mainly with the identification of fluid within the pleural cavity by the well known signs of massive, moderate, or slight opacity of the pulmonary field, mediastinal and cardiac shift to the opposite side, and a curved fluid level (Fig. 7*A*) changing slightly with the change in position of the patient. These features, together with a history of trauma, presuppose the presence of blood which must finally be confirmed by aspiration.

When hemothorax occurs in association with pneumothorax, and this is generally the rule, a horizontal fluid level changing freely with the position of the patient will be demonstrable (Fig. 7, *B* and *C*).

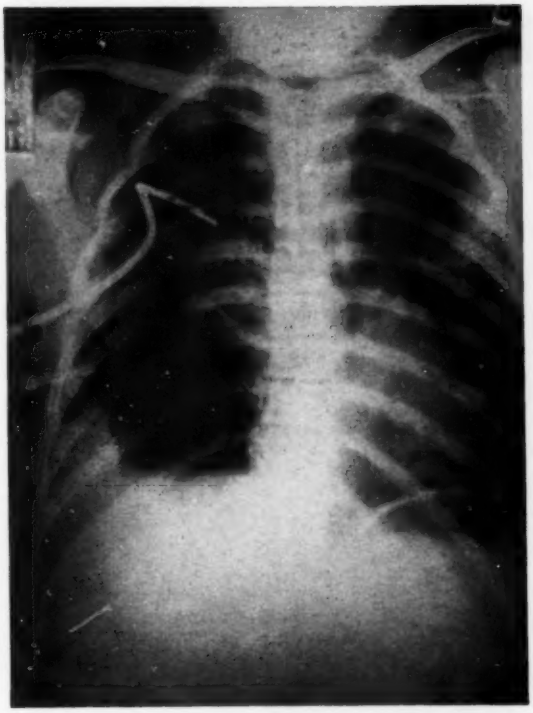

Fig. 6. Right-sided tension pneumothorax. The right lung is partially collapsed. The right midlobe is opaque. There is mild mediastinal shift, and a small accumulation of fluid obliterates the costophrenic sinus. There are mottled densities throughout the left pulmonary field attributed to parenchymal hematomas. Debrided fracture of right scapula and fracture of the third and fourth right ribs are also to be noted.

Loculated or Encapsulated Hemothorax. Loculated hemothorax is characterized by fibrous pleural bands enclosing accumulations of blood, with or without air, at various levels in the pleural cavity. The condition may be brought about by the deposition of fibrin from blood or, more particularly, by the fluid of a pleuritis which leads to an even greater fibrin deposit.[3] Septic hemothorax, by virtue of the associated increased inflammatory reaction, and clotted hemothorax, particularly after retraction and partial resorption have set in, are notably conducive to the formation of loculated hemothoraces. Moreover, the practice of air replacement, partic-

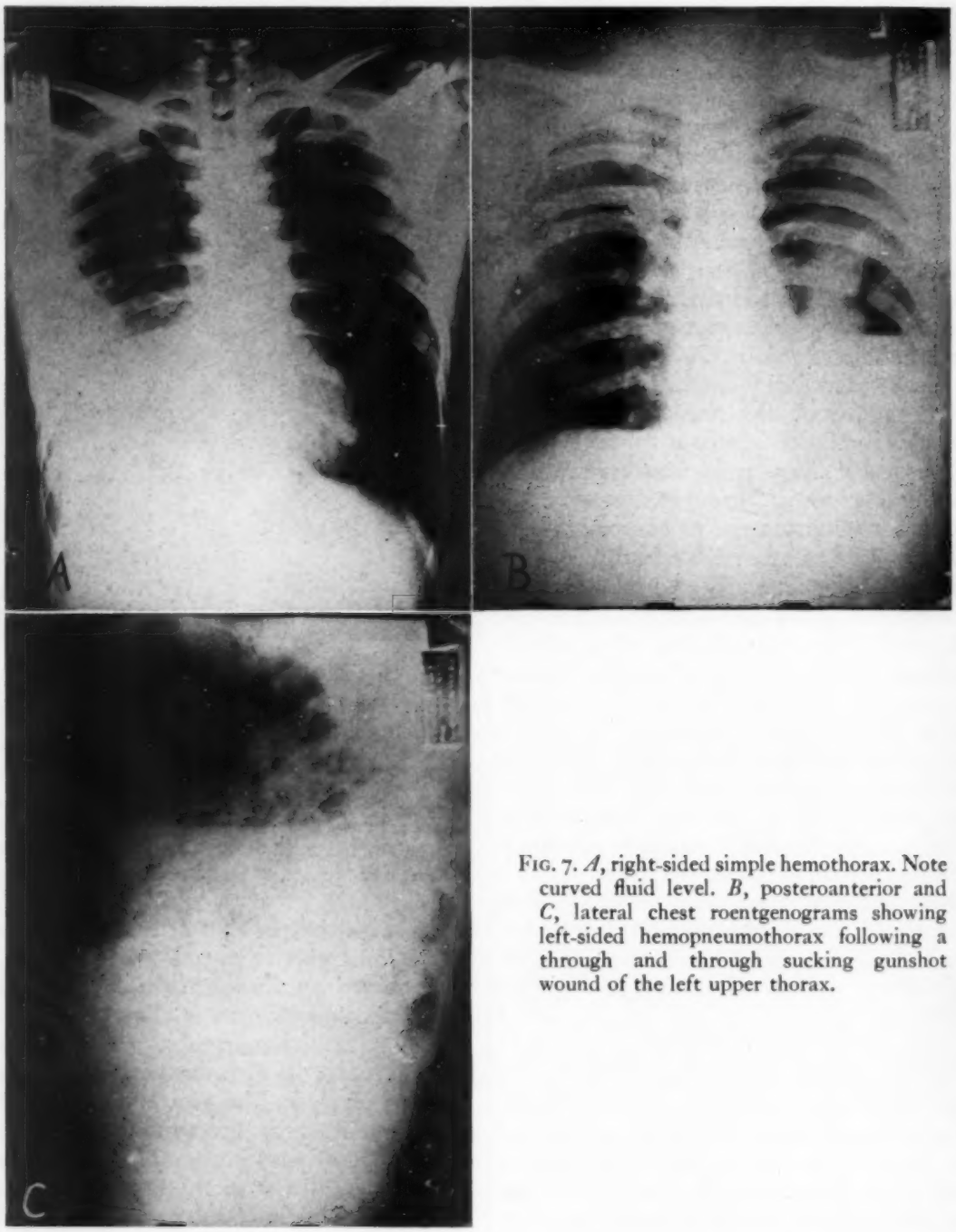

FIG. 7. *A*, right-sided simple hemothorax. Note curved fluid level. *B*, posteroanterior and *C*, lateral chest roentgenograms showing left-sided hemopneumothorax following a through and through sucking gunshot wound of the left upper thorax.

ularly after a first or second thoracentesis, serves to maintain pleural pockets.[4]

On the roentgenogram loculated hemothorax maybe represented as varying sized and irregularly distributed fluid levels (Fig. 8*A*). The loculations may occupy any portion of the main pleural reflection or there may be encapsulation within the interlobar fissures (8*B*). Moreover, loculated hemothorax may be identical with the features of clotted hemothorax, to be described later.

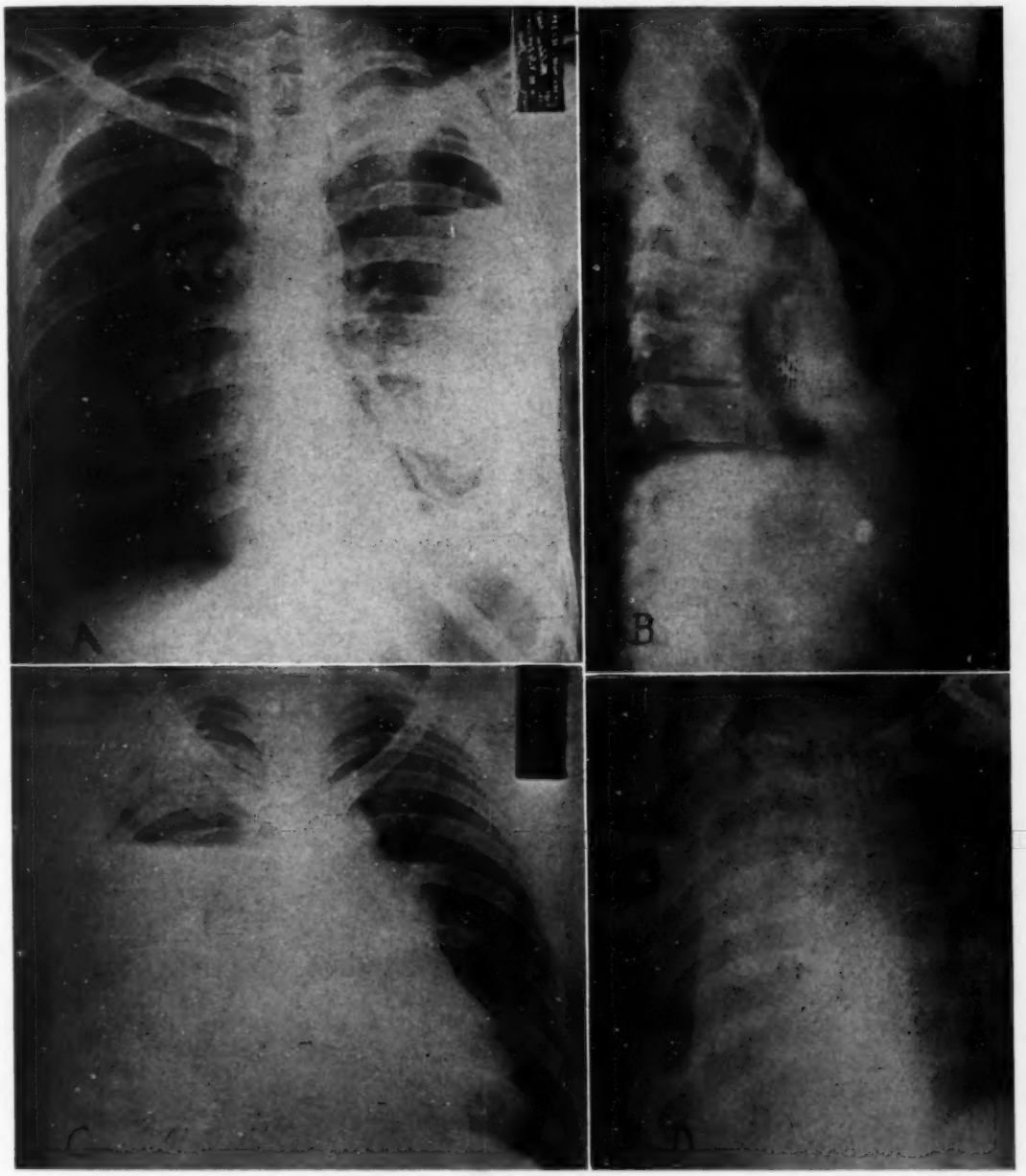

Fig. 8. *A*, left-sided loculated hemopneumothorax. Note the several discrete fluid levels within pleural cavity. This case cleared with repeated aspirations and remedial exercises. *B*, encapsulated interlobar fluid. Note subphrenic shell fragment, which had a transdiaphragmatic course. The fluid is encapsulated within the right main interlobar fissure. *C*, massive right hemopneumothorax (upright posteroanterior view). *D*, lateral recumbent roentgenogram (anteroposterior projection) shows several oculated areas of fluid in the same patient not discernible in routine view above.

In those instances where loculations are not satisfactorily demonstrable in the routine roentgenograms, anteroposterior roentgenograms taken in the lateral recumbent position may prove useful (Fig. 8*D*).

Accurate roentgen localization of loculated areas of fluid or air is important in realizing a conservative management of

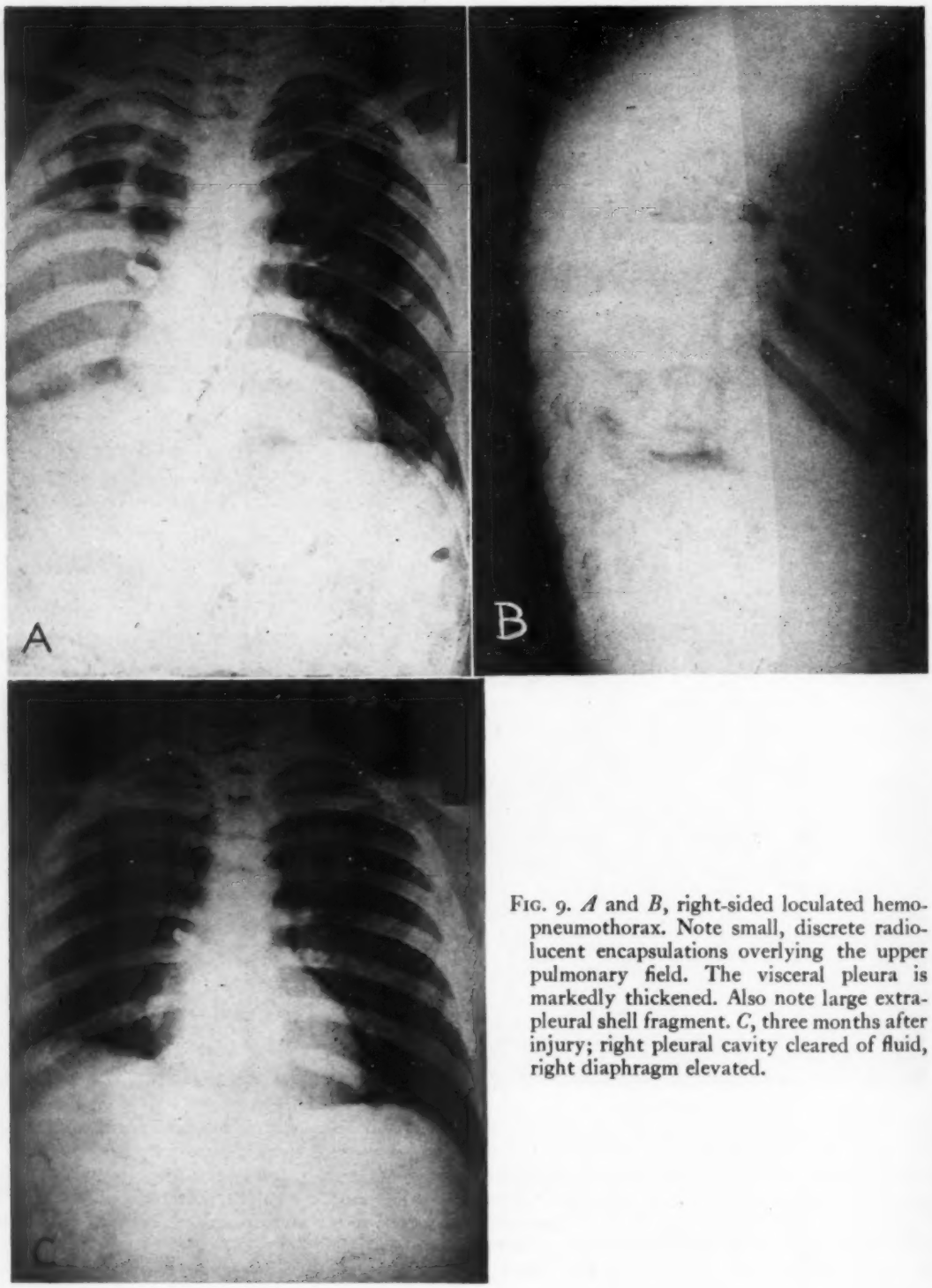

FIG. 9. *A* and *B*, right-sided loculated hemopneumothorax. Note small, discrete radiolucent encapsulations overlying the upper pulmonary field. The visceral pleura is markedly thickened. Also note large extrapleural shell fragment. *C*, three months after injury; right pleural cavity cleared of fluid, right diaphragm elevated.

such conditions by repeated thoracenteses and remedial exercises. The following case is an example of an infected loculated hemothorax which responded to conservative treatment.

This soldier, aged nineteen, sustained a pene-

trating shell fragment wound of the right chest on January 6, 1945. The missile entered at the region of the seventh rib at the costovertebral angle and lodged extrapleurally one rib below. The seventh rib was fractured at the entrance site. The wound was debrided the following day at an evacuation hospital where the patient received plasma, blood, a total of 300,000 units of penicillin and 18 grams of sulfadiazine, and two thoracenteses, which failed to yield fluid. The patient was evacuated to this hospital twenty days after his injury. A roentgenogram showed a loculated area of fluid (Fig. 9, *A* and *B*). Aspiration procured 330 cc. of chocolate colored pus which cultured gram-negative bacilli resembling Friedländer bacillus. Penicillin, 100,000 units, was placed in situ. This amount was also deposited with subsequent aspirations while sulfadiazine was administered parentally. A second thoracentesis several days later secured 300 cc. of thick yellow pus which proved sterile bacteriologically. A third aspiration procured 180 cc. of sterile, thick, purulent fluid, while a fourth aspiration, performed six weeks after injury, yielded 125 cc. of serous fluid. Roentgenoscopic follow-up at this time showed disappearance of the loculated area, obliteration of the right costophrenic angle by residual pleuritic thickening and limited diaphragmatic motion. At the time of discharge, three months following injury, the lung fields were entirely clear (Fig. 9*C*) and diaphragmatic motion was normal.

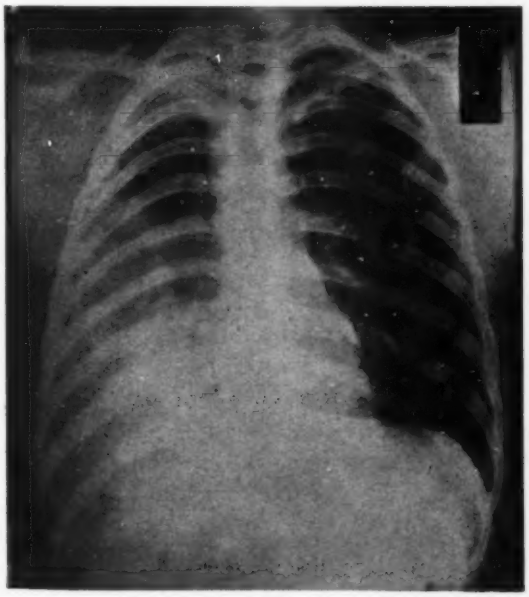

Fig. 10. Clotted hemothorax right side. Note the collapsed lung, the mottled densities, the fluid levels and the thickened pleurae.

Fig. 11. Clotted hemothorax right side. Note signs of traction evidenced by displacement of heart and mediastinum to the affected side.

Clotted Hemothorax. Clotted hemothorax occurs in about 4–6 per cent of cases with hemothorax.[2] The exact cause for clotting is not known. Infection is generally believed to play an important role in initiating the mechanism. Other factors suggested are the churning of the fluid imparted by respiratory and cardiac motion, the rapidity of bleeding, extensive parenchymal damage, the admixture of a pleural effusion, and the presence of a foreign body.

Roentgenologically, clotted hemothorax may assume a variety of appearances. There may be mottling, multiple fluid levels, densely thickened pleurae (Fig. 10) or signs of traction (Fig. 11).[7] These may appear alone or in combination.

Empyema. The incidence of pleural sepsis in battle casualties is given variously from 21.9 per cent in penetrating and 11.3 per cent in non-penetrating wounds[4] to 25–33 per cent.[2] Infection may result from the presence of foreign bodies, contaminants

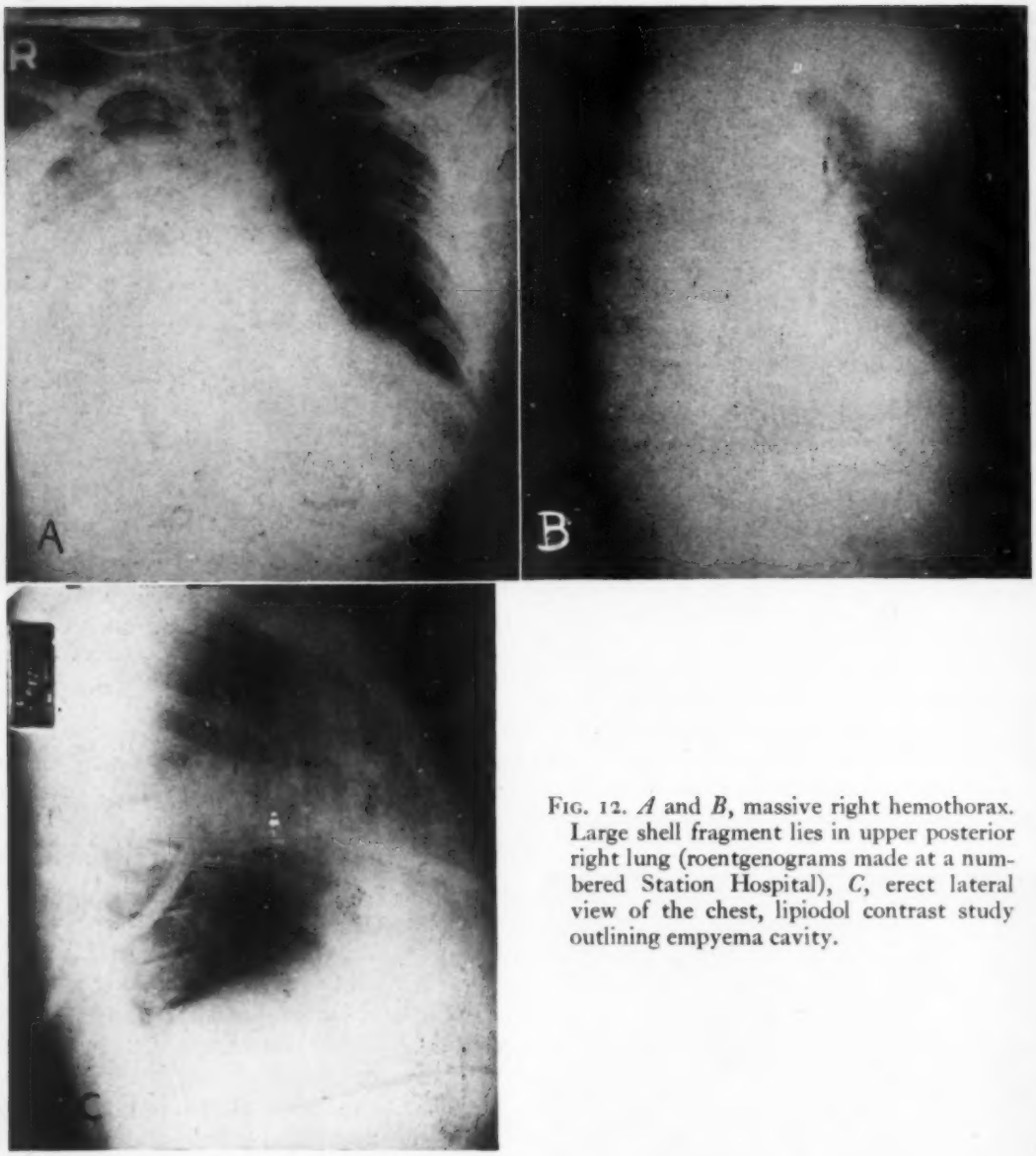

Fig. 12. *A* and *B*, massive right hemothorax. Large shell fragment lies in upper posterior right lung (roentgenograms made at a numbered Station Hospital), *C*, erect lateral view of the chest, lipiodol contrast study outlining empyema cavity.

brought in by foreign bodies, or from an improperly cared for wound; it may be blood borne or induced via devitalized lung parenchyma; it may occur postoperatively. By far the most frequently, infection is superimposed upon hemothoraces (Fig. 9) and pleural effusions, since the fluids serve as excellent culture media. Moreover, the greater the delay in aspiration, the higher the morbidity.[4] Chronicity or recurrence of empyema is attributable to inadequate drainage, the retention of foreign bodies, reinfection, undiscovered pus pockets, or osteomyelitis of adjacent bony structures.

The value of roentgenography in the diagnosis and localization of empyema is fully appreciated by the surgeon. Of special value are lipiodol contrast studies. These are employed as early as feasible to indicate the size, extent, and position of the empyema cavity, the most propitious site for the institution of drainage, and the presence or absence of sinus tracts. Lipiodol visualization, repeated at intervals, is also

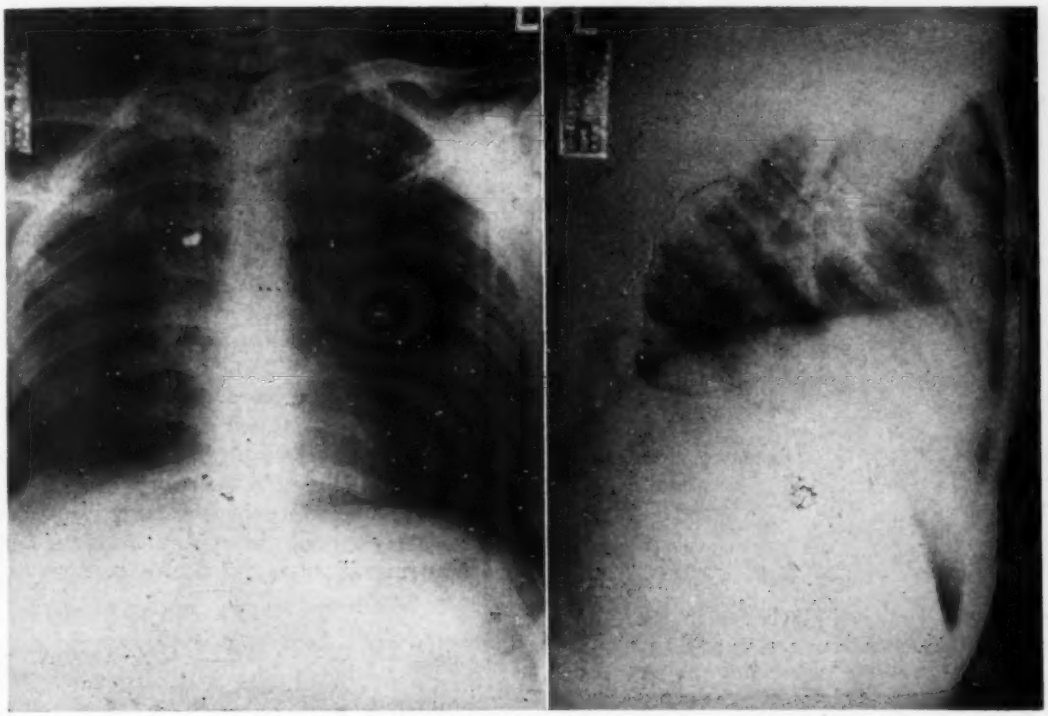

Fig. 13. Partial atelectasis right upper lobe. Mottled areas of increased density are noted. The trachea is retracted to the same side. In the lateral view the shell fragment was seen to be lying in the posterior thoracic wall.

useful to evaluate clearing and progress. The following case is cited as an example.

This patient, aged twenty-eight, was wounded in action on July 25, 1944, sustaining a penetrating, transdiaphragmatic wound. A 2 by 1.5 cm. irregular shell fragment, entering the right epigastrium, finally lodged in the right posterior thorax (Fig. 12, *A* and *B*). In the forward echelon, through an abdominal approach, lacerations of the right lobe of the liver and of the right diaphragm were sutured. Blood transfusions, plasma, oxygen, and chemotherapy were administered. Several thoracenteses proved futile.

Two weeks following his injury, the patient was admitted to this institution in a state of marked dyspnea. Clinical and roentgen findings denoted a massive hemothorax (Fig. 12*A*). Failure to again procure free fluid at aspiration gave weight to a suspicion of clotted hemothorax. Another transfusion was administered, and on the seventeenth day following injury a thoracotomy was performed. With the removal of an enormous amount of blood clot, fresh arterial bleeding from the markedly lacerated lung commenced. The vessel was ligated, the foreign body was removed, and the lung was sutured. Decortication of a thickened visceral pleura was then effected, and the thorax closed with intercostal drainage. About eighteen days after operation empyema became manifest. Primary aspirations followed by intercostal drainage and, later, rib resection, were instituted. Cultures were at all times negative. This was attributed to chemotherapy and the liberal use of intrapleural penicillin. Brewer tubes were placed in dependent positions (Fig. 12*C*) both posteriorly and anteriorly (at the site of the original thoracotomy) and negative pressure applied simultaneously through both tubes.

Atelectasis. Atelectasis is commonly found in association with thoracic wounds subsequent to compression phenomena, injury to the bronchial tree, and mucous plugs occurring as a postoperative complication. Its appearances on the roentgenogram vary from streak and plate-like densities to massive opacities. In the latter instance, there is retraction of the medias-

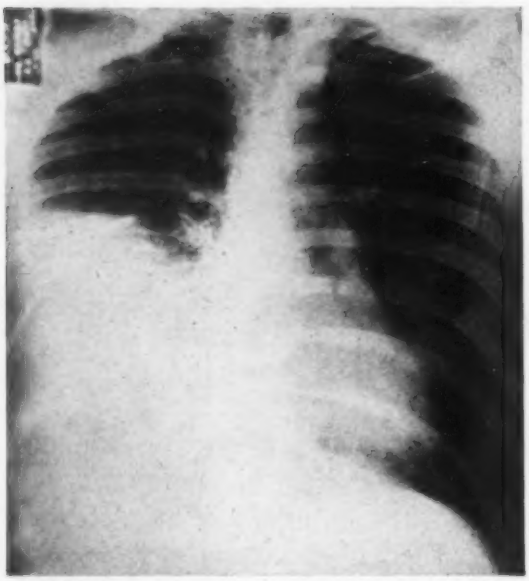

FIG. 14. Bronchopleural fistula demonstrated by injection of lipiodol into the sinus opening. Note the contrast substance overlying the hilar region. Note also extreme elevation of right diaphragm following phrenic crush.

tinum to the same side, narrowing of rib interspaces and elevation of the diaphragmatic dome. Figure 13 represents a case of partial atelectasis of right upper lobe following a penetrating shell fragment wound. The fragment, entering the right anterior second interspace, lodged in the posterior chest wall at the right costovertebral angle.

Bronchopleural Fistula. This complication occurs infrequently in thoracic wounds and generally follows in the wake of empyema, sinus tracts, or surgery. The following case is representative:

This soldier, aged thirty-eight, wounded in action on January 4, 1945, suffered a sucking chest wound which was caused by a shell fragment entering the right fifth interspace in the anterior axillary line and leaving at the right fifth interspace at the posterior axillary line. In addition to a sucking wound, the patient sustained fractures of the ninth, tenth and eleventh ribs, multiple extensive lacerations of the right diaphragm and lacerations of the liver and right lung. Resection of the ninth and tenth ribs and open thoracotomy were performed at the field hospital on the day of injury. The lacerations of the lung and diaphragm were repaired and the right phrenic nerve was crushed. Ten days after operation a thoracentesis yielded 200 cc. of purulosanguineous fluid. Another aspiration four days later procured 150 cc. purulosanguineous material and a large amount of air. At this time the sutures were removed from the chest wound. The anterior portion of the wound was separated and a large amount of pus was expressed. Upon admission to this institution, eighteen days after the day of injury, both purulent material and air were being expelled from the wound, the latter suggesting a bronchopleural fistula. Conservative treatment was followed. Penicillin and sulfa powder were employed locally. Lipiodol injected into the external opening gave evidence of the bronchopleural fistula (Fig. 14). The fistula headed spontaneously seven weeks after admission, at which time the lung fields showed normal illumination and the diaphragm some return of motion.

Blast Injury to the Chest. Concussion injuries to the lungs following waves of compressed air brought to bear upon the thorax occur in about 2 to 3 per cent of the cases. They may or may not be associated with damage to the chest wall or abdominal organs. The roentgenologic features of blast

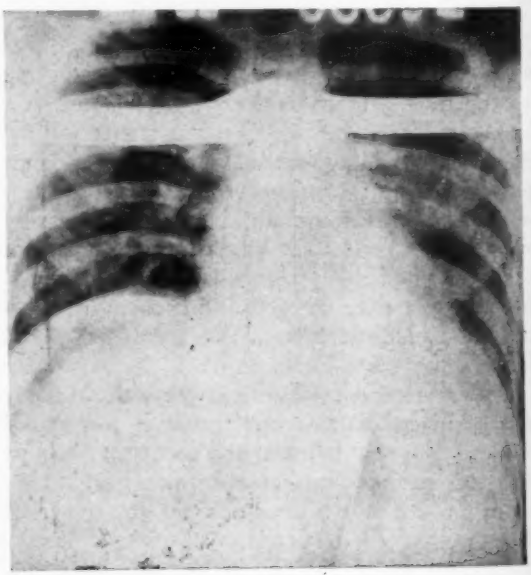

FIG. 15. Blast injury of chest. Note streak-like densities involving both pulmonary fields, most prominent at the bases (this roentgenogram was made at a numbered Field Hospital).

injury of the chest reflect the pathological findings; on autopsy[8] these are demonstrated as diffuse, bilateral, symmetrical areas of intrapulmonary hemorrhage mainly affecting the lower lobes. Coalescence may occur to represent areas of consolidation. Frequently the paravertebral gutters are involved, while subpleural hemorrhage is common. Figure 15 represents a case of blast injury of the chest in which bilateral, symmetrical mottling is a prominent feature.

Transdiaphragmatic Injuries. Thoracoabdominal wounds occur in 10 per cent of battle injuries of the chest and have a 25 to 40 per cent mortality rate.[2] It is not at all uncommon to receive patients who have sustained injuries to, and have survived operations upon the lung, diaphragm, liver, spleen, kidney and various portions of the gastrointestinal tract, in manifold combinations. Hepatopleural fistulae may be present or become manifest after admission. In our series alone we have been confronted with 4 such cases. Frequently the dia-

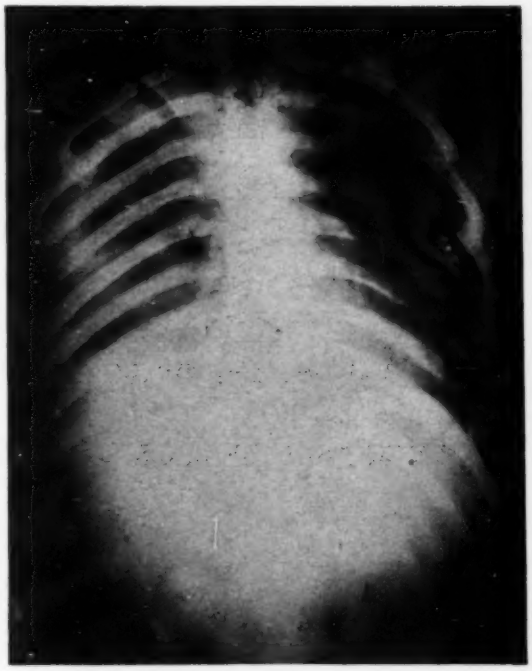

Fig. 16. Right pleural effusion secondary to penetrating wound of upper abdomen. Note the large abdominal fragment.

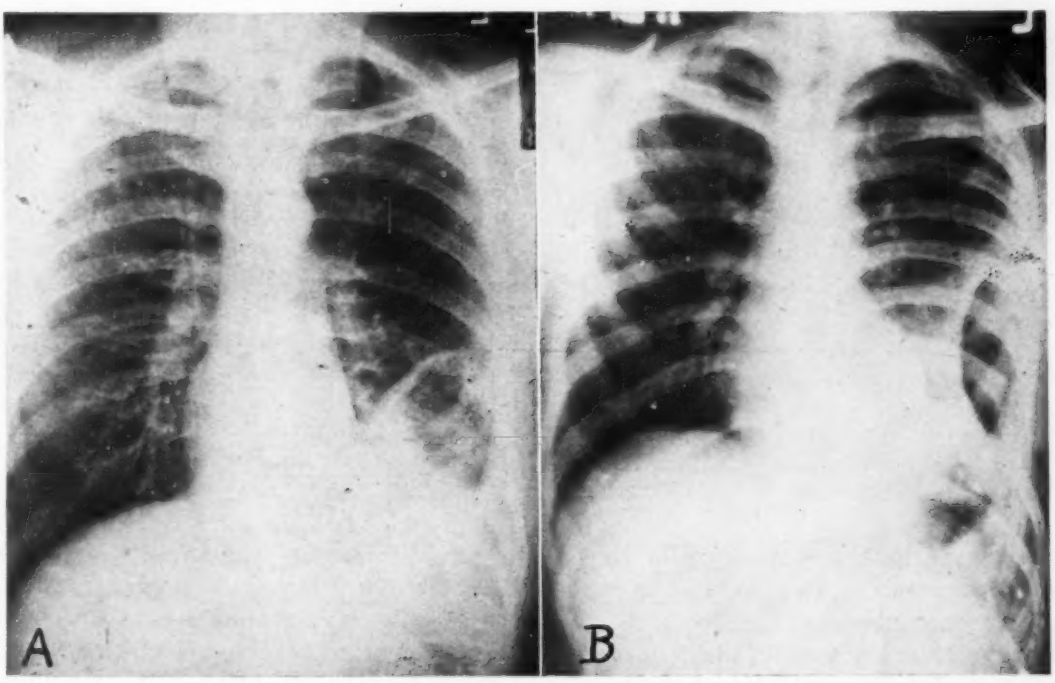

Fig. 17. *A*, traumatic diaphragmatic hernia. Note haustral configuration. *B*, sterile empyema cavity simulating *A*.

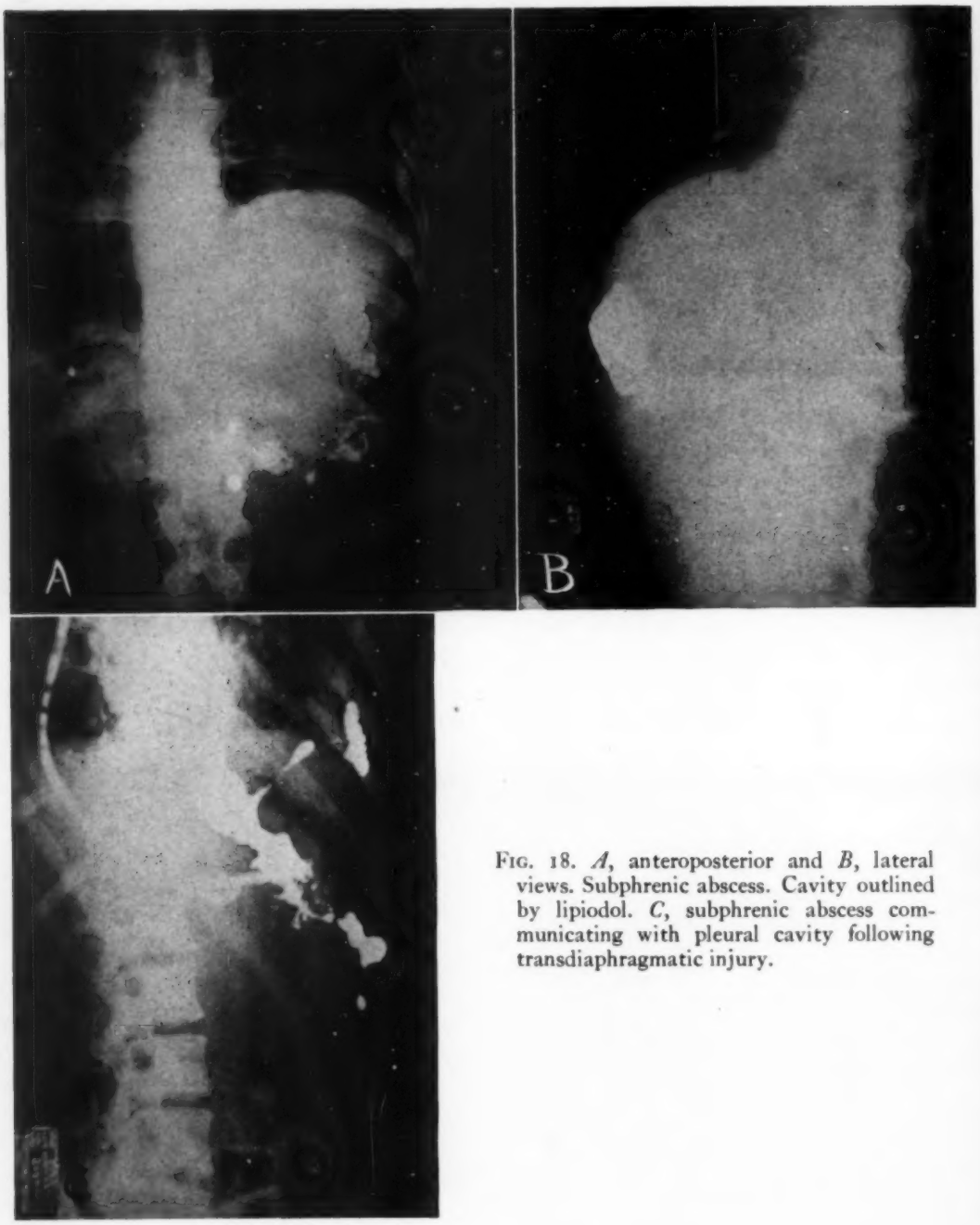

FIG. 18. *A*, anteroposterior and *B*, lateral views. Subphrenic abscess. Cavity outlined by lipiodol. *C*, subphrenic abscess communicating with pleural cavity following transdiaphragmatic injury.

phragm may be traumatized from below, without penetration, and result in extensive diaphragmatic pleurisy (Fig. 16).

In penetrating wounds of the diaphragm, immediate or delayed herniation of abdominal organs may follow. On the roentgenogram, diaphragmatic hernia must be differentiated chiefly from empyema cavity (Fig. 17*B*), loculated pneumothorax, interlobar fibrosis, lung abscess, eventration of the diaphragm, pulmonary cyst, and giant emphysematous blebs. Roentgenoscopy, barium studies and chest roentgenograms are utilized to establish a diagnosis. Figure 17 represents a diaphragmatic hernia of the colon following a through and through gun-

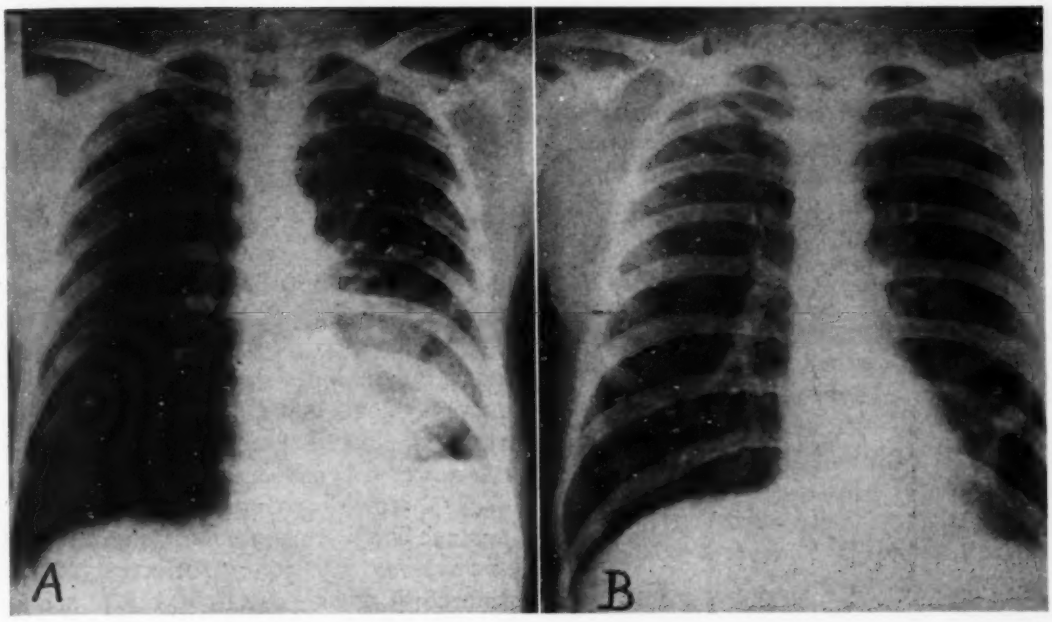

Fig. 19. *A*, hollow missile track traversing left lower pulmonary field. Surrounding opacity attributed to pneumonic consolidation. *B*, two months later. Except for a band of increased density which represents the site of the missile track, the left lower pulmonary field is clear.

shot wound of the lower left costal cage which lacerated the diaphragm.

Subphrenic abscesses constitute an additional interesting complication of transdiaphragmatic injuries, particularly when they communicate with pleural pockets. These cases are amenable to lipiodol contrast studies for outlining the cavities and for establishing the site of dependent drainage. Figure 18 shows 2 such cases, one demonstrating a subdiaphragmatic abscess without, and another with extension into the pleural cavity. In the first instance (Fig. 18, *A* and *B*) shell fragments entering the right tenth interspace in the posterior scapular line inflicted a hemopneumothorax and a laceration of the right diaphragm before lodging in the liver. In an evacuation hospital the wound was debrided, the foreign bodies were removed from the liver, the diaphragm was sutured, and the wound packed. Intercostal drainage was maintained for two days. Aspiration of 100 cc. blood-tinged fluid from the right chest was effected on the fourth postoperative day, and the packing was removed on the seventh. The patient was admitted to this institution fifteen days after injury. Thoracentesis procured 750 cc. of straw-colored fluid from the fifth interspace in the midaxillary line. Meanwhile, purulent material was discharging from the operative site. Lipiodol contrast studies were made; subsequently, dependent drainage of a subdiaphragmatic abscess was established and the patient made an uneventful recovery. In the second case (Fig. 18*C*) the patient sustained a transection of the transverse colon, laceration of the left diaphragm and lower lobe of the left lung following penetration of the eleventh interspace in the posterior axillary line by shell fragment. At the evacuation hospital a transverse colostomy and splenectomy were performed. The diaphragm was sutured and the phrenic nerve was crushed. Seventeen days following injury the patient was admitted to this institution where symptomatology indicative of subdiaphragmatic abscess became manifest.

Missile Tracks. Penetrating missiles occasionally leave roentgenologic evidence of their passage through lung tissue in the form of "tracks." These result from in-

filtration of blood along the course of the missile. Hodson[5] describes two types, namely, solid and air-containing tracks. The former is characterized by clear, linear or broad shadows leading from the entrance site to the site of lodgment or exit. These, when extensive, may sometimes require differentiation from pulmonary hematoma or abscess. Air-containing or hollow tracks appear as roughly parallel linear shadows with intervening air space which is maintained by a communication with the bronchial tree. When viewed end-on, these tracks, particularly if a fluid level is present, may resemble an abscess or tuberculous cavity.

Figure 19 represents a case of an air track which became infected and which was associated with an adjacent traumatic pneumonia.

On April 9, 1945, this patient sustained a sucking wound of the left chest and a hemothorax caused by a shell fragment entering the left tenth interspace in the posterior midscapular line. The wounds were debrided, the sucking wound was closed in layers, and water-seal intercostal drainage of the left thorax was maintained for three days. Four days later thoracentesis yielded 170 cc. of bloody fluid. The patient received transfusions and penicillin. Upon admission to this base hospital, twelve days after injury, the patient was expectorating thick blood, and, later, frankly purulent sputum. His breath had a strong, fetid odor. An area of dullness and bronchial breathing were clearly discernible over the lower left posterior chest. The patient's red blood count was 4,370,000 with 85 per cent hemoglobin, his white blood cell count was 18,250 with 95 per cent polymorphonuclear cells. The sutured wound over the tenth rib was infected. The sutures were removed and on separation of the wound purulosanguineous fluid escaped. A bronchopleural fistula then became manifest. The patient was placed on penicillin and sulfadiazine. The bronchopleural fistula healed spontaneously in three weeks. The sputum became less purulent, more mucoid, and in five weeks disappeared completely. Two months following injury roentgenograms showed the lung fields to be clear except for a band of increased density representing the previous hollow missile track (Fig. 19*B*).

SUMMARY AND CONCLUSIONS

Roentgenology plays an important part in the study and management of battle injuries of the chest. Additional views and positions should be utilized routinely, when feasible, to gather all possible information. Roentgenoscopic localization of fragments in relationship to or within the heart and great vessels, lungs, massive pleural effusions and chest cage, is of inestimable value to the surgeon. Traumatic entities encountered included: pulmonary contusion (parenchymal hematoma), simple, loculated and clotted hemothoracis, atelactasis, blast injury, bronchopleural fistula, missile tracks, transdiaphragmatic injuries, injuries to the bony thorax and subcutaneous emphysema. On follow up study, roentgenoscopy reveals extent of reëxpansion of pulmonary tissue, mobility of diaphragmatic domes, residual fluid levels and pleuritic adhesions.

The authors wish to express their appreciation to T/5 Earl W. Schenck for the photography concerned with this article.

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ACCESSORY LOBE OF THE LIVER AND ITS SIGNIFICANCE IN ROENTGEN DIAGNOSIS*

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IT IS not uncommon to visualize roentgenographically circumscribed soft tissue opacities at the base of the right hemithorax, continuous with the contour of the right hemidiaphragm. It is important to differentiate clinically significant lesions, such as parasternal hernia, or tumor of the diaphragm, from protrusion upward of an accessory lobe of the liver.

The following case illustrates the roentgen approach to this problem.

The patient, a white female, aged one year, was admitted to the Jewish Hospital on July 1, 1945, with an attack of watery diarrhea of approximately seventy-two hours' duration. Past history, including familial background, was entirely negative. Following administration of fluids, sulfones, and blood by vein, marked improvement occurred. She gradually became afebrile, appetite improved, but the swallowing of solid foods caused some difficulty.

FIG. 1

Because of this, a roentgen study of the esophagus was performed. Swallowing function was natural; the esophagus was negative.

Roentgenoscopic study of the chest revealed a sharply demarcated opacity in the anterior medial portion of the right hemithorax, occupying its lower half. Its superior surface was apparently continuous with the right hemidiaphragm; it moved synchronously with it. Roentgenographic study of the chest confirmed the roentgenoscopic observation. The soft tissue mass was demonstrated to be completely homogeneous (Fig. 1 and 2). The heart and mediastinal structures were slightly displaced to the left.

FIG. 2

The possibility that this might represent an accessory lobe of the liver was considered, but it was decided that a parasternal hernia should first be excluded. There was no evidence of herniation of the stomach or small intestine. To exclude an omental hernia, a barium enema

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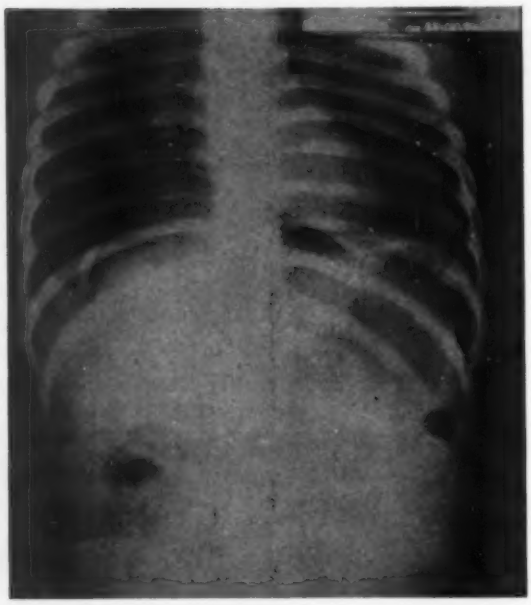

FIG. 3

study was done. There was no evidence of upward displacement of the colon, as demonstrated in the presence of omental hernia by Robbins.[5]

In order to prove that this represented an accessory lobe of the liver, a diagnostic pneumoperitoneum was performed under roentgenoscopic guidance. Fifty cubic centimeters of air was injected, and anteroposterior and right lateral roentgenograms in the upright posture were made (Fig. 3 and 4). The pneumoperitoneum was visualized beneath the right hemidiaphragm, separating the diaphragm from the mass. The mass was demonstrated to be continuous with the superior surface of the right lobe of the liver. It apparently represented an accessory lobe of the liver, which had invaginated a portion of the right hemidiaphragm. Follow-up examinations revealed no change.

DISCUSSION

This case graphically demonstrates the roentgen approach that should be adopted in an analysis of this type of lesion.

Parasternal hernia is much more common on the right, appearing as a sharply demarcated opacity in the right lower lung field adjacent to the heart. Its density may be uniform if it contains only omentum or fluid filled intestine. If colon is present within the hernia, haustrations may be visible. Barium meal and enema studies are essential for recognition. If only omentum is present, there are varying degrees of upward and medial displacement of the right hemicolon, with upward displacement of the stomach.

Tumors of the diaphragm are rare, with less than 20 cases reported. The majority are benign, appearing as lipomas, fibromas and myofibromas. The malignant tumors are fibrosarcomas or fibromyosarcomas. These sarcomas grow rapidly, metastasizing to lungs, pleura, and bones. When these tumors are circumscribed, they are continuous with the diaphragm in contour and mobility, and can best be demonstrated by a combined pneumothorax and pneumoperitoneum.

Accessory lobe from the superior hepatic surface similar in size to the above described lesion is relatively uncommon. Less than 15 cases have been reported. Their recognition is important, since they are clinically benign. They can best be demonstrated by pneumoperitoneum. If an inflammatory mass such as hepatic abscess

FIG. 4

is causing the protrusion, adhesions are usually present, preventing the entry of air into the subdiaphragmatic space. The diagnostic problem involved is well demonstrated by the case reported by Roques and Sohier.[6] A Senegalese male, who had a low grade fever, and an eosinophilia of 11 per cent, demonstrated a sharply circumscribed mass in the anteromedial portion of the right lower hemithorax. Pneumoperitoneum demonstrated its continuity with the liver, and a diagnosis of echinococcus cyst was made. Exploratory operation, however, demonstrated a circular accessory lobe of the superior surface of the liver, 10 cm. in maximum diameter. It is not possible to absolutely differentiate accessory lobe of the liver from projecting benign tumors such as angiomata. These tumors are usually only a few millimeters in diameter, however, The frequently observed anteromedial bulge of the right hemidiaphragm probably represents a variation in contour of the superior hepatic surface. The roentgen demonstration of this group of lesions should therefore include roentgenoscopy, and posteroanterior and lateral roentgenograms of the chest, barium meal including small intestinal study, barium enema, and finally pneumoperitoneum.

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THE ROENTGEN FINDINGS IN ACUTE PANCREATITIS*

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INTRODUCTION

THE pancreas is so oriented in the upper abdomen that, despite the fact that it cannot be visualized roentgenologically, it does when diseased induce changes in the neighboring organs which may frequently lead to a correct roentgen evaluation of the underlying abnormality. The abnormalities which may occur on the roentgenogram are so manifold and often so bizarre that it is incumbent upon the radiologist to be thoroughly conversant with both the anatomy of the pancreas and the pathology of acute pancreatitis. Unarmed with this knowledge one could not reasonably hope to put together the varied patterns which may be manifested.

Acute pancreatitis is by no means rare, yet the clinical diagnosis is rather infrequent, and all too often the diagnosis is made either during surgical exploration, or at the autopsy table. Undoubtedly this is due to a lack of awareness of the pancreas as the exciting factor in acute abdominal conditions. Because roentgen examination plays such an important role in the diagnosis of the acute abdomen, we present this discussion in order to focus attention on the roentgenologic features of acute pancreatitis.

ANATOMY OF THE PANCREAS

The strategic anatomic relationships of this gland readily lend credence to the known varied signs and symptoms of acute pancreatitis. It is an organ which normally has the appearance of one of the larger salivary glands, being somewhat lobulated and firm. The normal weight varies between 2¼ and 3½ ounces, and it is usually 5 to 6 inches in length. It occupies a transverse position in the upper abdomen on the posterior wall retroperitoneally, and extends from the right, where its head is in contact with the concavity of the duodenum, to the left where its tail is in contact with the visceral surface of the spleen. Some of the more detailed relations of the pancreas are shown in Table I.

As a rule, there are two excretory ducts of the pancreas—the main duct of Wirsung, and the accessory duct of Santorini. The duct of Wirsung commences in the tail of the pancreas from the union of the smaller branches and gradually increases in size as it progresses through the pancreas from the left to the right. It then passes downward and backward through the neck to the head where it lies near the posterior surface and is in relation to the common bile duct, beside which it enters the duodenum in an oblique fashion 2 to 3 inches below the pylorus. Just after this point they unite to form the ampulla, or "diverticulum of Vater," which is their common discharge point into the duodenum.

The accessory duct of Santorini drains a large area of the head of the pancreas. It usually anastomoses with the duct of Wirsung near the neck, and increases in size as it approaches this point. Opie[22] has found that in more than half of the cases the accessory duct is obliterated or so constructed as to render it incapable of assuming the function of the larger duct should the latter become occluded. Rienhoff and Pickrell[24] recently reported anatomical studies on 250 dissected cadavers, and compared their results with previous studies. In 24 per cent of the cases there was no junction of the pancreatic and bile ducts. In 37 per cent with contiguous ducts the septum terminated 1 to 2 mm. from the apex of the common orifice; in 2 per cent of the cases the main pancreatic duct was fibrotic; in 18 per cent with an ampulla, the diameter of the latter exceeded the average width of the orifice so that a complete block of the papilla could result in a communicating system. These findings were discussed in relation to the various reported etiologic factors in pancreatitis.

INCIDENCE OF PANCREATITIS

Acute diseases of the pancreas are by no means rare. In various reported series it has

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been estimated that they constitute anywhere from 0.3 per cent to 0.95 per cent of the general autopsy series.[13,32] Milder forms are undoubtedly more common than is generally recognized.

Lagerlöf,[13] and Eliason and North[12] state that in their series of cases acute pancreatitis is more common in women than in men—a statement which is at variance with those of Weiner and Tennant,[32] Whipple,[34] and Muller.[20] Various figures have ranged from a preponderance of 79 per cent in males to 59 per cent in females.

The age incidence is usually given as between twenty and sixty years, but there have been cases reported in almost all age groups.[12,13]

It seems to be the consensus that acute pancreatitis is more common in the obese, heavy eaters and drinkers than it is in the thin and light eaters.[13,20,34]

TABLE I

ANATOMIC RELATIONS OF THE VARIOUS PORTIONS OF THE PANCREAS

Part	Right	Anterior	Left	Posterior
Uncinate process	1. Vessels along third portion of duodenum 2. Duodenum			
Head	1. Concavity of the duodenum	1. Commencement of transverse colon 2. Small intestine	1. Superior mesenteric vessels	1. Inferior vena cava 2. Left renal vein 3. Aorta 4. Common bile duct 5. Right renal vessels
Neck	1. First portion of the duodenum 2. Pylorus (when stomach is distended)		1. Superior mesenteric veins 2. Splenic veins	1. Portal vein
Body		1. Stomach 2. Inferiorly: a. duodenojejunal flexure b. jejunum	1. Left flexure of colon	1. Aorta 2. Origin of superior mesenteric artery 3. Pillars of diaphragm 4. Splenic artery and vein 5. Left kidney 6. Renal vessels 7. Left suprarenal capsule
Tail		1. Inferiorly: a. Left flexure of colon	1. Lower part of inner surface of the spleen	

ETIOLOGY OF ACUTE PANCREATITIS

There is probably no other disease entity in which there is so much disagreement as to etiology, both experimentally and clinically. The literature on the subject is too voluminous to review completely. However, some of the more important etiologic factors are summarized below (after Lewison):[14]

I. Infectious Origin
   - A. Spread to pancreas through lymphatics[12,18,20,31]
   - B. Through blood stream[12,14,18]
   - C. Through pancreatic or bile ducts[7,21,34]
   - D. Direct extension from local foci, such as kidney, gallbladder, etc.[7,13,21]
   - E. Activation of bacteria and toxins in normal gland[14]

F. Complication of other diseases such as mumps, typhoid, scarlet fever, diphtheria, small pox[17]

II. Non-infectious Origin
- A. Reflux into the pancreatic ducts of:
  - 1. Bile due to:
    - a. Stone in ampulla of Vater[22]
    - b. Spasm of sphincter of Oddi[1,20]
    - c. Edema of ampulla of Vater[14]
    - d. Diverticulum of duodenum[18]
  - 2. Duodenal contents[13]
- B. Erosion of peptic ulcer[12]
- C. Obstruction of pancreatic ducts due to:
  - 1. Epithelial metaplasia of duct epithelium[25]
  - 2. Miscellaneous: tumors, either malignant or benign, stricture, edema, ascaris worms, pancreatic calculi[13,18,25]
- D. Trauma
  - 1. Surgical[18]
  - 2. Anesthesia[18]
  - 3. Accident[18,20,25]
- E. Vascular accidents
  - 1. Embolus[18,27]
  - 2. Thrombosis[18,27]
  - 3. Cardiac decompensation with vascular congestion[18]
- F. Autolysis of pancreas[14]

III. Chemical
- A. Arsenic[18]
- B. Alcohol[18,20,32]

IV. Combination of Factors[14]

Biliary tract disease is the most commonly suggested factor in the etiology of pancreatitis. Dragstedt, Haymond and Ellis[10] have estimated that it constitutes the etiologic agent in approximately 60 per cent of all the cases. Opie[22] advanced the theory that the lodgment of gallstones in the ampulla of Vater producing a reflux of bile into the pancreatic duct was the cause of pancreatitis. Since this theory was advanced, much work has been done which is some cases bears out the "common channel theory," and in some instances disputes it. For example, Mann and Giordano[15] have shown that in order to obtain reflux of bile into the pancreatic ducts, it is necessary to inject it with a pressure of 1,000 mm. of water, which is considerably higher than the secretory pressure of the biliary tract. Harms (quoted by Rienhoff and Pickrell)[24] has reported that during digestion in the unanesthetized dog, the secretory pressure of the pancreas is greater than that of the biliary tract. These findings, to some, refute the theory advanced by Opie,[22] but even these authors admit that the "common channel theory" may be operative in the causation of a small percentage of the cases. Eggers[11] has stated that ligation of the pancreatic duct would produce pancreatitis only if the animal were in an active phase of intestinal digestion. Wangensteen, Leven and Manson[29] were able to produce pancreatitis in 15 of 31 cats by ligating the sphincter of Oddi.

Archibald[2] showed that the injection of sterile bile into the pancreatic duct was capable of producing only pancreatic edema, and he was able to produce pancreatic necrosis only if the injected bile was infected. Rich and Duff[25] in their series of cases suggested that pancreatitis was due to a metaplasia of the ductile epithelium which resulted in an obstruction leading to rupture of the acini and ductules distal to the obstruction. This damage to the acini and ductules behind the obstruction permits the escape of pancreatitic juices into the interstitial tissues of the pancreas, and in the event that the escaping juice is rich in tryptic ferments, a pancreatic necrosis results. They also state that rupture of thinned out acini and ductules behind the obstruction is most likely to occur during periods of increased pressure which may follow the ingestion of a large meal or alcohol. They were of the opinion that the epithelial metaplasia bears no relation to inflammation.

Weiner and Tennant,[32] in a study of 4,000 autopsies, found 38 cases with acute pancreatic lesions, and 97 with chronic lesions. They thought that in 25 of the acute cases alcohol played an etiologic role. In only 6 of the cases of acute pancreatitis was there evidence of extrahepatic biliary tract disease. In 51 individuals dying during acute alcoholic episodes, 25 showed acute pan-

creatic lesions and 2 showed chronic lesions. There were 41 cases of chronic alcoholism of which 19 showed pancreatic lesions, all of them being chronic. Of 51 cases of periportal cirrhosis, 25 showed pancreatic lesions, almost all of them being chronic. Of the 343 cases of extrahepatic biliary tract disease, there were only 6 cases of acute pancreatic lesions, and 21 cases of chronic pancreatic lesions. Their conclusions were that the incidence of biliary tract disease is increased in pancreatitis, but that the incidence of pancreatitis in biliary tract disease was only slightly, if at all, increased over that observed in the general autopsy series.

It has been shown that the injection of almost any material into the pancreatic ducts is capable of producing pancreatitis.[13] The great amount of clinical and experimental material in the literature indicates the complexity of the problem, and it is probably safe to say that there is not always a single factor operative in the causation of acute pancreatitis. It is probable that in many cases there may be a combination of factors which are either directly or indirectly responsible for this disease entity.

SIGNS AND SYMPTOMS

Acute pancreatitis is usually characterized by a sudden onset of continuous severe epigastric pain, nausea, vomiting, distention, and collapse. However, the same findings are frequently present in other abdominal conditions so that more often than not the diagnosis is biliary tract disease, a perforated viscus, or acute intestinal obstruction. Any patient, with or without a previous history of intermittent epigastric discomfort, who is suddenly seized with a severe epigastric pain, followed by vomiting and collapse must be suspected of having acute pancreatitis.

The pain has been described as "severe," "excruciating," "knifelike," "unbearable," "agonizing," etc. In some patients it is intractable even to large doses of opiates.[18]

Moynihan[19] has described the pain as being a formidable catastrophe, which is by far the most severe of all pains that the human body can suffer. Lewison[14] agrees with this statement, but believes that pain of such severity occurs only in the hemorrhagic and necrotic types of pancreatitis. According to him, the pain present in the edematous form is much less severe.

Wangensteen[30] states that in any acute lesion of the abdomen accompanied by severe pain and signs of collapse, but without actual shock, and not attended by intestinal colic, with most of the findings confined to the upper abdomen, pancreatic necrosis must be considered. The pain may be localized to one or both groins, epigastrium, or supraspinous area.[8] Morton[18] reports that in his series of cases 55 per cent had the pain localized to the epigastrium, 32 per cent had pain at the costovertebral angle, 11 per cent had pain in the right and left upper quadrants, 1 per cent had generalized pain, and 1 per cent had pain in the right lower quadrant. De Takáts and Mackenzie[28] reported 30 cases all of which had severe pain. In 10 of the cases the pain was in the right upper quadrant and right shoulder, and 7 had pain in the epigastrium radiating to the left.

Most of the patients have vomiting or retching which may be intractable, but usually there is little actual emesis. It is more persistent than that occurring in perforation of a viscus and is almost never feculent. There may or may not be nausea accompanying the vomiting or retching. Morton[18] reports that in a few of his cases blood was observed in the vomitus on several occasions.

Lewison[14] in his series of cases, which he divided into four classes of pancreatitis, noted that vomiting was present in 71 per cent of the cases, and that nausea appeared to be even more common. Abdominal symptoms, though present in all patients, varied in intensity and location, and he found that there was no relation between the degree of tenderness and the type of pancreatic lesion present.

The temperature is often at shock levels and according to Cope,[8] may be as low as

95° F. Morton,[18] however, states that it may be low, normal, or slightly elevated. Some have stated that the pulse rate is disproportionately higher than the temperature, which is at variance with the observations of Dragstedt, Haymond and Ellis.[10]

A few of the cases show cyanosis which is usually best seen on the face and extremities, but this is not a constant finding. Occasionally some of the patients have complained of dyspnea.

Epigastric tenderness is a constant finding, but spasm of the abdominal muscles is a variable feature. When spasm is present it is usually of the type seen in biliary tract disease.[30] Borborygmus and abdominal distention are not uncommon features but, as a rule, gas pains are conspicuously absent. This absence of gas pains serves as an important differential point between acute pancreatitis and obstruction of a viscus. Constipation is the rule.

Within a short time following the onset of the process, there may be a tender palpable mass occupying a transverse position in the upper abdomen. Coincidental with this, jaundice may be noted and has been reported in as high as 50 per cent of all cases.[18] It is due to the involvement of the common duct by the inflammatory process. Because of the extravasation of blood ecchymoses may occur over the flanks or around the umbilicus, but this does not take place, if at all, until the third or fourth day of illness.

The physical findings reported in the literature have varied considerably, and apparently the main point of agreement is the sudden onset of severe pain in all cases. It is also stressed that the pain commonly follows a hearty meal or a drinking bout.

LABORATORY FINDINGS

De Takáts and Mackenzie,[28] reporting 30 cases, present the laboratory findings. Most of these 30 cases showed the effects of dehydration. Four had white blood cell counts of less than 4,000, four had white blood cell counts of greater than 30,000, but more commonly the counts ranged between 15,000 and 20,000. Eleven of the cases had albumin in the urine, 9 had granular and hyaline casts, 4 had sugar, 2 had acetone and diacetic acid, and 2 had red blood cells. In 1 case the blood sugar was 75 mg. per 100 cc. of blood. The urea nitrogen in 1 case was 23.3 mg. and the non-protein nitrogen was 48.4 mg. per 100 cc. of blood. A sugar tolerance test on 1 patient showed totals of 122, 227, 272, and 219 mg. of sugar per 100 cc. of blood at 0, ½, 1, and 2 hours, respectively, with 4.9 gm. of sugar being excreted into the urine. In 1 patient peritoneal fluid was examined and was found to contain 10 per cent bile. Cultures for bacteria were negative.

Krotoske (quoted by de Takáts and Mackenzie[28]) has stated that a high fasting blood sugar level is present in pancreatic necrosis and only a diffuse purulent peritonitis will give values comparable to those seen in pancreatitis. The value must be over 160 mg. of sugar per 100 cc. of blood to be of significance. Morton[18] has also reported the occurrence of high blood sugar values in some of his patients with one of them having a fasting sugar level of over 400 mg. per 100 cc. of blood. He states that the blood sugar was uncontrollable with insulin.

Of much interest has been the use of the serum amylase and lipase, and urinary diastase determinations in the diagnosis of acute pancreatitis. Whipple[33] stated that he preferred the serum lipase determinations as a diagnostic aid. Skoog[26] has reported 3,000 diastase determinations in other acute abdominal conditions and he concluded that a negative test, provided it was done within twenty-four to thirty-six hours after the onset of the disease, rules out the presence of a pancreatic lesion. Polowe[23] has more recently reiterated the value of the serum amylase test, and states that the presence of a high amylase value almost always means pancreatic disease. However, he points out that in some cases of high intestinal obstruction, before intestinal damage ensues, there may be a significant absorption of these enzymes, producing an elevated serum amylase level. The serum amylase value may decline

sharply within seventy-two hours after the onset of the disease so that after this time the serum amylase test is of little diagnostic value. Hence the test must be performed early in the course of the disease. Polowe[23] also states that a moderate increase may be associated with a grave prognosis, and that a normal or low value almost always excludes the possibility of pancreatitis. A slightly elevated amylase value may be indicative of secondary involvement of the pancreas due to a primary pathological process elsewhere. In his paper he lists 15 different processes which may produce an elevation of the amylase value, and 14 processes which may result in a lowering of the value. He has also devised a simplified method for quick determination of the serum amylase value which should be of great use.

According to Lagerlöf[13] the indirect tests which were devised before the direct methods of examination (i.e., amylase, diastase, and lipase determinations) are not to be relied upon. These tests include the nucleus test of Schmidt and Kashiwada, starch hydrolysis and sugar absorption, stool enzyme determinations, digestion and absorption of fats and proteins, and Cammidge's reaction. He himself has devised the secretin test which he has used to advantage. Loewe's test (described by Moynihan[19] and Cope[8]) has been of value in a certain number of cases.

PATHOLOGY OF PANCREATITIS

Waring and Griffiths[31] have described the development of acute hemorrhagic pancreatitis as first beginning with swelling in the head, tail, or body. This is then followed by the development of a serous peritoneal exudate. The swelling which is firm is rapidly followed by softening and hemorrhage which may infiltrate between the layers of the transverse mesocolon, into the root of the mesentery, into the retroperitoneal tissues, or lesser or greater sacs. Fat necrosis also appears at this time. The pancreatic juice is then mixed with blood which results in the foul-smelling, chocolate-colored exudate which is usually seen. The presence of infection is then manifested as a peritonitis develops. Since they believe that all pancreatitis is caused by infection, the peritonitis is explained on this basis.

Large areas of the pancreas become necrotic and are sloughed, either through an operative wound, or as in one case they report, spontaneously into the rectum. They believe that the development of suppurative pancreatitis is dependent upon the amount of hemorrhage and the site of the hemorrhage. If the hemorrhage is confined to the capsule of the pancreas then the development of infection leads to suppurative pancreatitis.

It must be realized that the above events occur within a matter of hours.

Lewison[14] has divided acute pancreatitis into four groups: (1) acute edematous pancreatitis, with or without fat necrosis; (2) acute hemorrhagic pancreatitis, with or without fat necrosis; (3) acute gangrenous pancreatitis, with or without fat necrosis; and (4) acute suppurative pancreatitis, with or without fat necrosis. In each instance the pathological findings are those of the descriptive terms used.

Of interest are the vascular lesions described by Rich and Duff.[25] In their series of cases of hemorrhagic pancreatitis they found a peculiar type of vascular necrosis. The adventitia of the vessels (both arteries and veins) appeared condensed and some contained leukocytes or necrotic cells. The media showed swelling of the muscle fibers and they were sometimes separated by clear spaces or by pink staining fluid. The nuclei of the cells were shrunken, or pyknotic and often karyorrhectic. The internal elastic lamina had lost its undulations and appeared swollen. Individual fibrils were split off leaving a frayed appearance.

It was these vascular lesions, which they were able to reproduce experimentally by use of trypsin, that they believed explained the hemorrhage seen in hemorrhagic pancreatitis. Whether or not hemorrhage will occur in any one case, they think, depends upon the proximity of blood vessels and amount and activity of the pancreatic ferments released into the substance of the

pancreas. Should there be no vessels near, or should the amount be small and/or the activity low there, only a small area of fat necrosis results.

Pathologically, the changes within the pancreas run the whole gamut from simple edema to complete necrosis.

Various authors have disputed whether there is actually a bacterial agent present, and there is even greater dispute as to whether the bacteria that are found at times are of etiologic significance or if they are merely secondary invaders. De Takáts and Mackenzie[28] believe that in at least a good many instances they are merely contaminants from the intestinal flora.

Dragstedt, Haymond and Ellis[10] found *B. welchii* organisms in the pancreatic lesions of many of their experimental animals and suggested that the same organisms may in some way contribute to the toxic appearance of some of the patients.

In one of de Takáts and Mackenzie's[28] cases a pleural empyema was found, and they observed that pleural effusion was not uncommon.

Areas of fat necrosis appear throughout the peritoneum. These are apparently due to the pancreatic steapsin breaking down the fats into fatty acids and glycerin, with the subsequent formation of calcium soaps. Waring and Griffiths[31] report that this occurs in all cases and is most common in the region of the transverse colon, root of the mesentery, and omentum. In a few instances it has been observed in the pericardial and extrapleural fat of the chest. Apparently this is due to the pancreatic ferments circulating in the blood stream. Some of the changes observed in the chest may be due to infectious or other agents gaining entrance into the chest through the wealth of tiny lymphatic openings in the diaphragms.

ROENTGEN FINDINGS IN ACUTE PANCREATITIS

Case[6] in his excellent paper has called attention to several roentgen findings associated with pancreatitis. Most of these findings have been duplicated in our cases. Case[6] has described elevation of the stomach and transverse portion of the duodenal bulb, enlargement of the duodenal loop, collection and stasis of contrast material in dependent portions of the duodenum (atony), and defects in the shadow contours in the region of the duodenojejunal flexure, and beyond that, normal filling of the jejunal loops. These changes have been ascribed to changes occurring within the head of the pancreas, and are due either to mechanical or inflammatory processes.

Other findings which he describes are sensitivity to pressure in the region of the pancreas with fullness between the stomach and transverse colon, limitation of excursion of the left diaphragm, pleural exudates and interlobar cicatrices especially on the left, ill defined left psoas shadow, pressure indentations in the stomach and duodenum, coarsely feathered duodenal folds, abnormal gaseous distention of the colon, sometimes paralytic ileus with fluid levels, and an indistinct shadow in the region of the pancreas.

Metheny, Roberts and Stranahan[16] have likewise reported a group of cases in which similar changes were noted, and their report tends to corroborate the findings of Case.[6] They also mention the frequency with which a gas bubble is to be seen in the atonic duodenal bulb.

While we would agree with most of the changes recorded by others, in our experience we cannot claim agreement concerning the stress placed upon atony of the duodenum. True, we have found in some an atonic bulb filled with gas, but more often our cases have shown rather persistent spasm of the duodenal loop associated in some with contour changes (Fig. 1), but in others with no such changes (Fig. 2). One case showing particularly marked narrowing and spasm of the duodenum actually had an annular pancreas as well as pancreatitis. This was previously reported by one of us.[4]

Also, we would emphasize the anatomical and physiological changes seen in the proxi-

mal jejunum, where there is frequently marked persistent spasm of some segments along with apparent or real dilatation of others, and associated with this there is likewise a pronounced and persistent coarsening of the folds. The picture is indeed a graphic one attesting to the alterations in

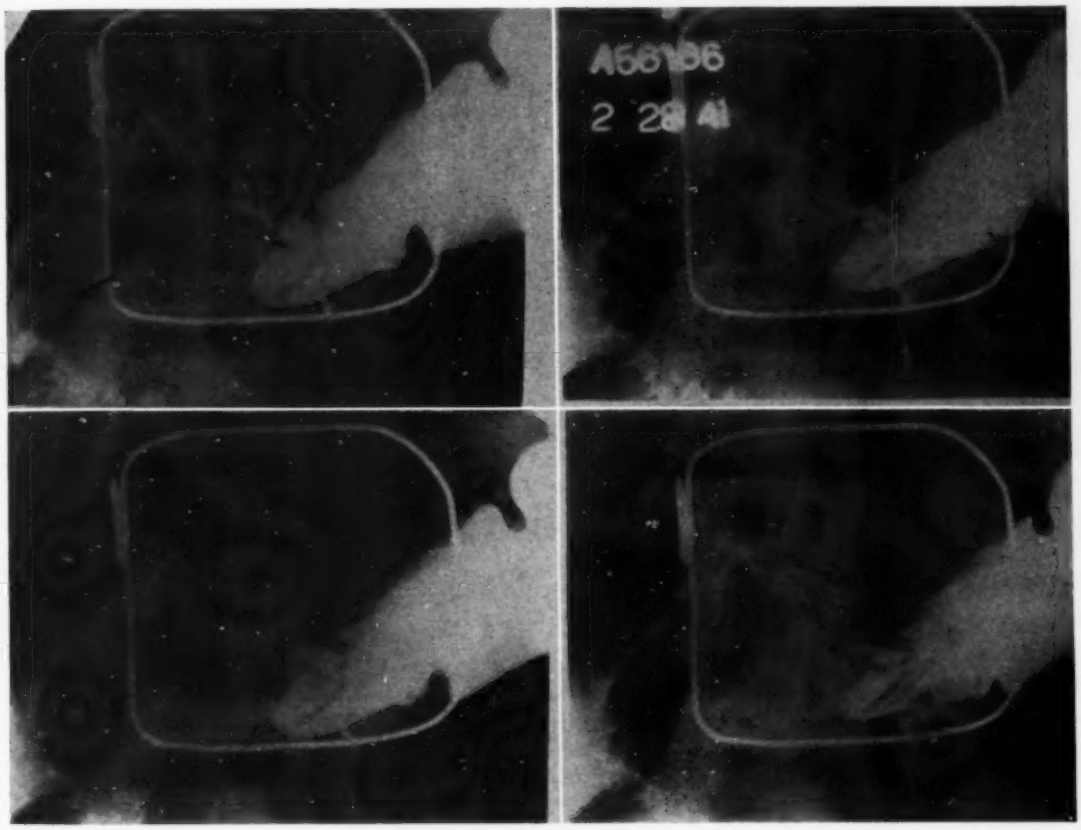

Fig. 1. Serial roentgenograms showing enlargement of the duodenal loop associated with persistent spasm. Arrow points to inverted "3" deformity.

the mucosal relief as well as in the peristaltic function (Fig. 3).

In keeping with this pattern of spasm are the findings reported by one of us[3] demonstrating constant areas of spasm in the colon at the mid-transverse portion and the splenic flexure, both regions being zones of direct contact with the pancreas. There was marked ileocecal spasm secondary to the extension of the fat necrosis in this area. The rest of the colon was of normal caliber or perhaps slightly dilated, but the picture was not that of general atony. The patient's obstipation was undoubtedly due to deranged neuromuscular function.

This same case showed roentgenologically an obliteration of the normal visceral detail, but the roentgenogram was characterized by a mottled appearance. The nodular shadows of soft tissue density varied from 1.0 to 3.0 cm. in diameter, and they coincided with the numerous areas of fat necrosis found at autopsy.

From the anatomic relations of the various portions of the pancreas, one might expect the changes visible roentgenographically to be varied, depending upon which portion is involved. The head and uncinate process being in contact with the concavity of the duodenum, commencement of the transverse colon, small intestine, common

FIG. 2. Note persistent spastic contraction of descending duodenal segment. No enlargement of the loop was present. The bulb may be slightly enlarged and displaced upward.

bile duct, and several blood vessels may cause various changes due, as Case[6] mentions, either to mechanical or inflammatory processes due to changes within the pancreas. These changes may be summarized as follows: If there is appreciable enlargement of the head of the pancreas the duodenal loop may be widened and the valvulae conniventes flattened, and the inverted "3" sign of Frostberg may be evident (Fig. 1). As already described there may be spasm of the duodenal loop (Fig. 4 and 5). Along with these changes there may be a pressure defect or marked spastic contraction of a portion of the transverse colon (Fig. 6), and some of the small intestine in the region may show similar changes; that is, alternate areas of spasm and dilatation. The foramen of Winslow may become sealed off isolating the lesser omental sac where large collections of fluid may displace the stomach, as reported by one of us.[3] The common bile duct may become obstructed resulting in enlargement of the gallbladder and liver.

The neck of the pancreas is in close proximity to the bulb and pylorus, so that the bulb may be lifted or distorted in disease of the pancreas. Also, the stomach may be pushed up or forward depending upon its anatomical relationship in any particular patient. The stomach may also show a filling defect which is difficult at times to differentiate from an intrinsic lesion.

The body of the pancreas is in relationship with the stomach, duodenojejunal flexure, small intestine, left flexure of the colon, pillars of the diaphragm, left kidney, and aorta and other blood vessels. The possible stomach changes are those mentioned before. The intestinal changes are similar to those which may occur elsewhere, and are so uniformly present that they are an important part of the over-all picture. The left colic flexure may show the same changes induced in the transverse colon when the head is involved. An extremely important relationship is the diaphragmatic one, for many cases of pancreatitis will show evidence of fluid at the left base

FIG. 3. This case shows the varied small intestinal pattern characterized by spastic segments, slightly dilated segment, and definite mucosal fold coarseness.

(Fig. 7, *A* and *B*), undoubtedly due to the inflammatory changes occurring in the diaphragm, or beneath it. Any abdominal case which develops fluid at the left base had best be suspected of having pancreatic disease until proved otherwise. Another frequent roentgen change is obliteration of the left psoas and renal shadows due to edema or inflammation of the soft tissues as a result of the pancreatic disease.

The tail of the pancreas contacts the diaphragm, splenic flexure of the colon, and spleen, so that changes similar to those described above may be manifested.

It may not be amiss to here mention the role of duodenal diverticula in pancreatitis. In the absence of any acute abdominal

Fig. 4

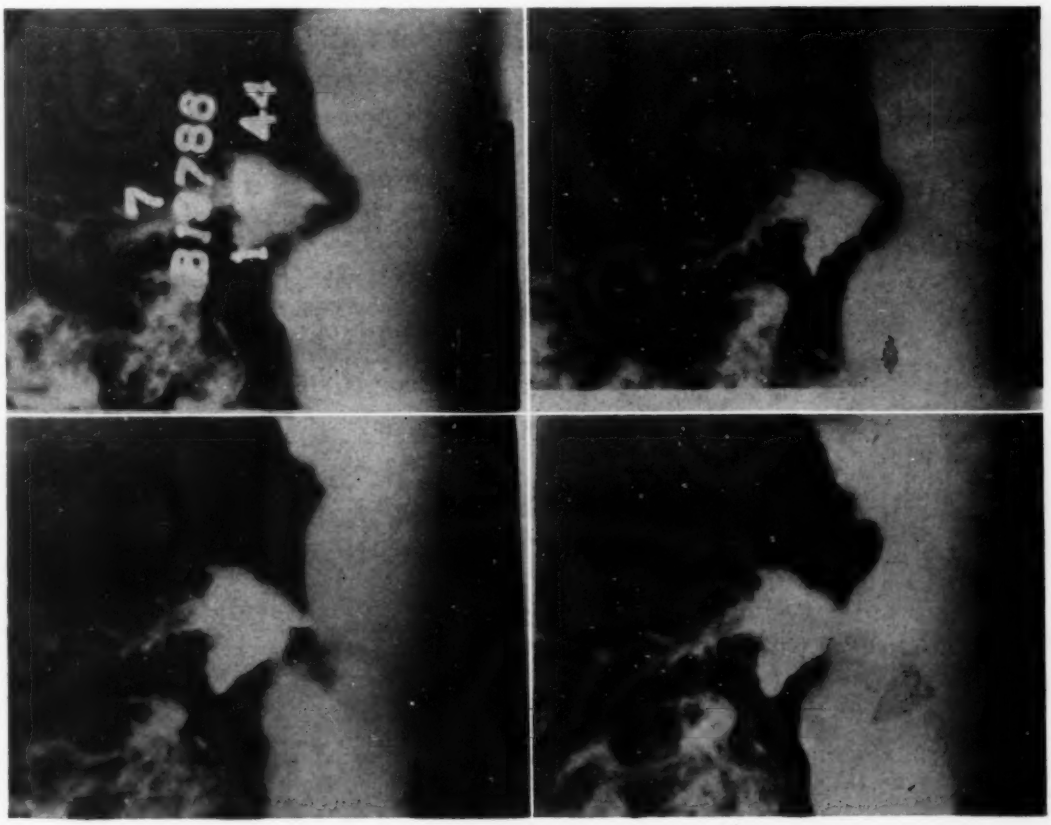

Fig. 5

Fig. 4 and 5. Two different cases which demonstrate the persistent spastic changes present in the duodenal loop without any obvious pressure deformity.

FIG. 6. The colon is well filled and the spastic contraction of the proximal portion of the transverse segment is well illustrated. There is perhaps some dilatation at the splenic and hepatic flexures. There is also spasm in the pelvic segments.

complaints, the finding of a duodenal diverticulum is not particularly alarming. However, should one be seen in a patient with acute upper abdominal symptoms findings associated with the diverticulum may be of great importance. Particularly should one determine the mobility of the diverticulum, whether it fills well and empties well, the presence of spasm or irritability, the presence of any defects in the contour of the diverticulum, and displacement for if some or all of these aspects are present it must be remembered that a diverticulum which is buried within the pancreatic substance may, when inflamed, lead to pancreatic disease. Hence, once the diverticulum is found and thought to be abnormal, other changes secondary to pancreatitis should be investigated.

It is evident from the foregoing discussion that the radiologist must be well informed concerning the anatomical relationships of the normal pancreas before he can hope to intelligently evaluate the many roentgen aspects of acute pancreatitis. On the other hand, when armed with this knowledge he may often contribute materially to the solution of a difficult clinical problem.

CONCLUSIONS

1. The anatomy of the normal pancreas is reviewed and the clinicopathological findings of acute pancreatitis discussed.

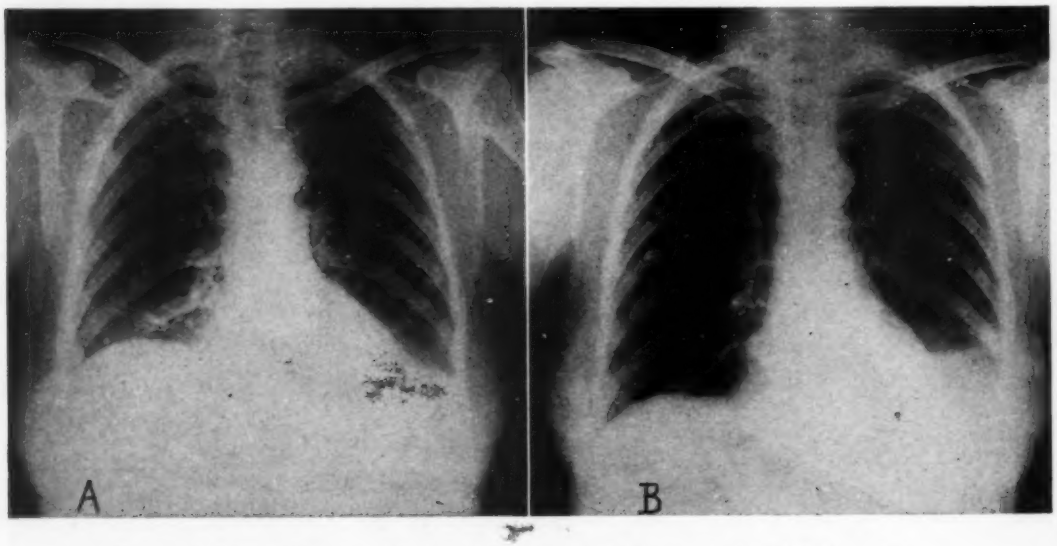

FIG. 7. (*A*) There is no evidence of fluid at the left base, but in (*B*) (roentgenogram taken nine days later) there is obvious effusion.

2. The roentgen findings in acute pancreatitis are described with emphasis placed upon the regional abnormalities induced because of the strategic anatomical relationships of the pancreas.

3. Stress is laid upon the spastic changes seen in the gastrointestinal tract, changes frequently accompanied by zones of apparent or real dilatation.

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DEXTROCARDIA WITH COMPLETE SITUS INVERSUS

CASE REPORTS

By LIEUTENANT COLONEL SAMUEL RICHMAN
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DEXTROCARDIA with complete situs inversus was found in 3 instances of approximately 15,000 roentgen examinations of the chest made at this hospital. The ages of the patients were twenty, twenty-one and twenty-three years. They presented no symptoms referable to the heart and had no difficulty in carrying out

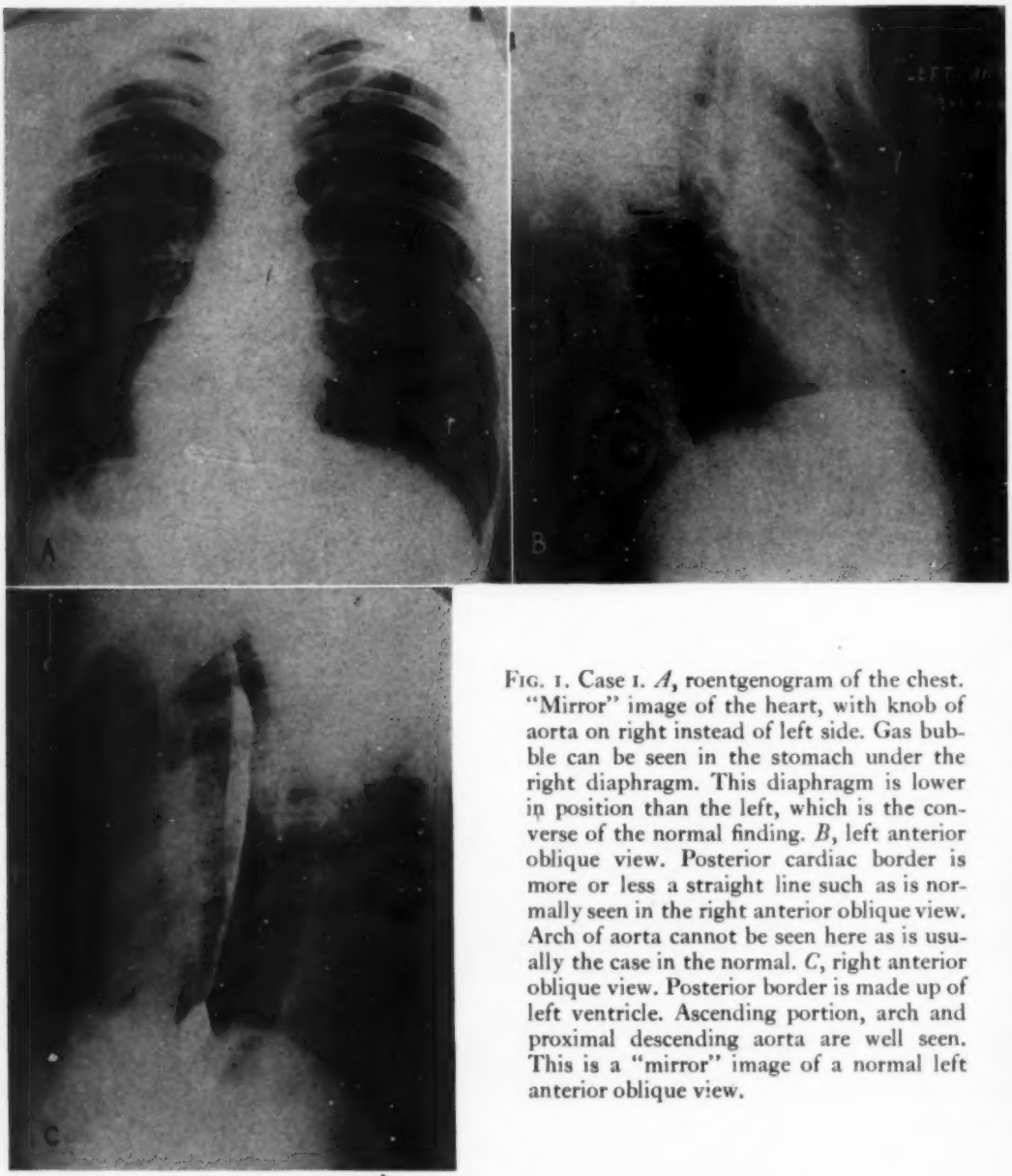
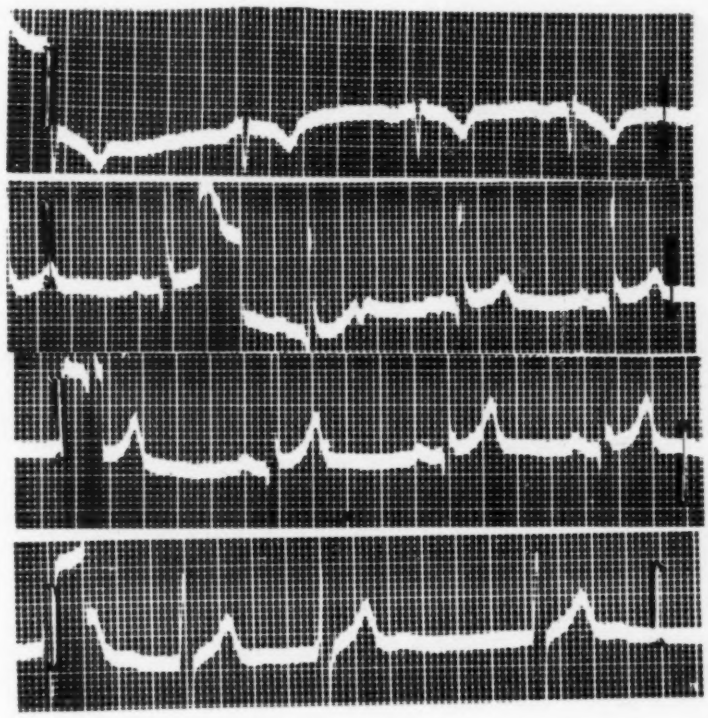


FIG. 1. Case 1. *A*, roentgenogram of the chest. "Mirror" image of the heart, with knob of aorta on right instead of left side. Gas bubble can be seen in the stomach under the right diaphragm. This diaphragm is lower in position than the left, which is the converse of the normal finding. *B*, left anterior oblique view. Posterior cardiac border is more or less a straight line such as is normally seen in the right anterior oblique view. Arch of aorta cannot be seen here as is usually the case in the normal. *C*, right anterior oblique view. Posterior border is made up of left ventricle. Ascending portion, arch and proximal descending aorta are well seen. This is a "mirror" image of a normal left anterior oblique view.

the routine Army tasks. Two of these men were unaware of the existence of the abnormality, while one had been told about it when he was in high school. No abnormal physical findings aside from the location of position as compared to the left in each instance. Barium examination was done in 2 of the patients and here again a true "mirror" image of the stomach, small and large intestine was obtained. The clinical signifi-



Rate (per min.) 70 Auricular 70 Ventricular 70 PR interval 0.18
Rhythm Sinus arrhythmia.
P waves P-1 inverted.
QRS complexes 0.09. QRS-1 inverted. ST-2 and ST-3 elevated but within normal limits.
T waves T-1 inverted.
Interpretation Total inversion of Lead I characteristic of congenital dextrocardia.

FIG. 1. Case. 1. *D*, electrocardiogram.

the left border of the heart were recorded in any of the individuals.

Characteristically, the heart in each of the 3 cases was seen as a "mirror" image with the left ventricle pointing to the right. The right hemidiaphragm was lower in

cance of knowing beforehand on which side of the abdomen the cecum and appendix are located can be readily appreciated in acute abdominal episodes, therefore the patient should be instructed to report his anomaly to the physician.

FIG. 2. Case II. Barium meal examination shows stomach on right side with duodenal bulb to left of spine.

FIG. 3. Case III. Barium enema examination shows cecum and ileum on the left with descending colon and sigmoid on the right side.

SUMMARY

Three cases of dextrocardia with complete situs inversus, in 15,000 successive examinations of the chest, are reported.

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FRACTURE OF A SESAMOID BONE OF THE THUMB

REPORT OF A CASE

By LIEUTENANT COLONEL DAVID L. JELLINGER
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FRACTURE of a sesamoid bone of the thumb is thought to be sufficiently rare to justify reporting. Only one case has been reported in the United States. None has been reported in the American roentgenological literature. Three cases have been reported in the British literature. As far as could be determined, no other cases have been reported.

The first case recorded appears to have been in 1919 by Pye[1] in his textbook, "Surgical Handicraft." The text reads:

> The skiagram shows the result of a sprain of the thumb sustained by a medical student during a football match, and it will be noticed that the sesamoid bone is fractured; the thumb was forcibly hyper-extended with this result. As far as we know, this is the only case of fractured sesamoid recorded.

The second case was reported by Streatfeild and Griffiths[2] in 1934, and was of a fifteen year old schoolgirl who gave a history of jumping a "horse" in a gymnasium class during which her right hand "got left behind" and she twisted her thumb. She was seen one hour later and had swelling and severe pain in the region of the metacarpophalangeal joint of the thumb. Roentgen examination showed a fractured sesamoid at the head of the metacarpal.

The third case was reported by Sinberg[3] in 1940 and was that of a student nurse aged twenty-two, who stumbled over a chair and in trying to save herself from falling struck the right thumb violently against an iron bedstead. She felt immediate pain over the head of the first metacarpal and could not use the right thumb for the next few days. Local tenderness, swelling, and a moderate degree of ecchymosis were present. Roentgenographic examination revealed a fracture of the radial sesamoid. Pain on use of the thumb persisted for several months and then gradually disappeared. A roentgenogram taken twenty-five months after the injury showed complete bony union.

The fourth case was reported by Scobie[4] in 1941 and was that of a man aged sixty-two, who was lightly holding the steering wheel of his car in his right hand, the thumb being uppermost, when he collided with a stationary car. The force of impact, though not great, was transferred through the steering column and wheel to the base of the thumb. He experienced sudden severe pain at the site, followed by stiffness and some swelling of the thumb. A roentgenogram revealed no fracture or dislocation of the thumb, but the sesamoid bone on the ulnar side showed a crushed appearance.

The case reported here is that of a military motorcycle policeman, aged twenty-six, who gave a history of having lost his balance while on a motorcycle, and of having fallen on his outstretched hand, striking the ground with a good deal of his weight borne on his left hand. The

FIG. 1.

Fig. 2.

Fig. 4.

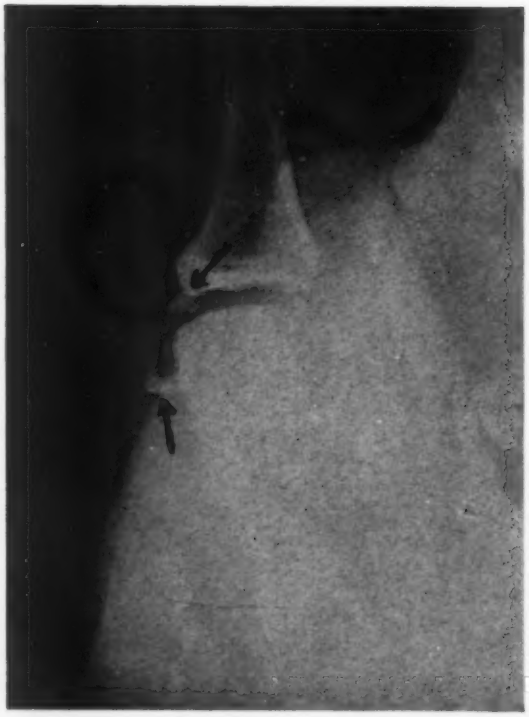

Fig. 3.

Fig. 5.

next day he complained of pain, swelling, and stiffness of the thumb, and was sent to the hospital for roentgen examination. On physical examination, he had tenderness and swelling over the palmar aspect of the metacarpophalangeal joint, extending into the muscles of the thenar eminence. He could not move his thumb actively, and passive movement caused pain.

Routine and tangential roentgenograms of the thumb (Fig. 1, 2, 3, 4, and 5) were made and these revealed a fracture of one of the sesamoids with about 1 cm. distraction between the fragments. Some rotation of the distal fragment was present. The tangential exposures were made so that the shadows of the fragments would be projected away from the overlying metacarpal and proximal phalanx.

DISCUSSION

In all the cases reported, direct trauma was the etiological factor in the fractures.

If the fragments are in close apposition, it is necessary to differentiate between a bipartite sesamoid and a fracture. In the case reported here, the question of bipartite sesamoid did not arise because of the distraction between the fragments, the rotation of the distal fragment, and the irregular jagged margins of the fractured surfaces. It may be necessary to watch for the development of callus as confirmatory evidence of a fractured sesamoid.

Scobie, in his case report, states that the frequency with which injuries of the thumb occur without a Bennett fracture or other recognized bony or articular derangement is so well known that the term "staved" thumb is a household expression in England. In this country the injury is probably better known as a "sprained" thumb. In any patient presenting himself with a sprained or "staved" thumb, the possibility of a fractured sesamoid should be borne in mind. Since in routine views the sesamoids of the thumb may be overlapped by the adjoining metacarpal and phalanx, tangential exposures are desirable for better demonstration of the sesamoids.

Symptoms may last a few days to several months. Either the radial[3] or ulnar[4] sesamoids may be fractured.

As far as treatment is concerned, immobilization has resulted in good function.

SUMMARY

A case of fractured sesamoid of the thumb is reported and four previously recorded cases are reviewed.

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DOSAGE CALCULATIONS IN RADIUM THERAPY

AN EXHIBIT

Prepared by EDITH H. QUIMBY, Sc.D.
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THIS EXHIBIT WAS PREPARED, BY REQUEST, FOR THE SECOND INTER-AMERICAN CONGRESS OF RADIOLOGY, IN HAVANA, 1946. IT WAS ALSO SHOWN AT THE MEETINGS OF THE AMERICAN ROENTGEN RAY SOCIETY AND THE RADIOLOGICAL SOCIETY OF NORTH AMERICA IN THAT YEAR. THE EDITOR OF THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY FELT THAT THE PUBLICATION OF THIS MATERIAL BY PHOTOGRAPHS OF THE ORIGINAL CHARTS WOULD SERVE A USEFUL PURPOSE.

NO NEW MATERIAL IS INCLUDED IN THESE CHARTS. THE AUTHOR HAS ENDEAVORED TO COLLECT BASIC INFORMATION AND ARRANGE IT IN SUCH A MANNER AS TO EMPHASIZE SALIENT POINTS. FUNDAMENTAL CHARTS AND TABLES ARE PRESENTED, WITH EXAMPLES OF THEIR USE. FOR DETAILED DISCUSSIONS, THE FOLLOWING REFERENCES ARE SUGGESTED:-

PATERSON & PARKER. DOSAGE SYSTEM FOR GAMMA RAY THERAPY. BRIT. J. RADIOL. 7, 592, 1934.
PATERSON & PARKER. DOSAGE SYSTEM FOR INTERSTITIAL RADIUM THERAPY. BRIT. J. RADIOL. 11, 252 & 313, 1938.
QUIMBY. PHYSICAL FACTORS OF INTERSTITIAL RADIUM THERAPY. AMER. J. ROENTG. & RADIUM THER. 33, 306, 1935.
QUIMBY. SPECIFICATION OF DOSAGE IN RADIUM THERAPY. AMER. J. ROENTG. & RADIUM THER. 45, 1, 1941.
QUIMBY. DOSAGE TABLE FOR LINEAR RADIUM SOURCES. RADIOLOGY 43, 572, 1944.
STENSTROM, QUIMBY, AND PENDERGRASS. REPORT OF RESEARCH AND STANDARDIZATION COMMITTEE OF AMERICAN RADIUM SOCIETY. AMER. J. ROENTG. & RADIUM THER. 43, 118, 1940.

(THANKS ARE DUE TO DRS. PATERSON AND PARKER FOR PERMISSION TO INCLUDE THEIR MATERIAL IN THIS EXHIBIT)

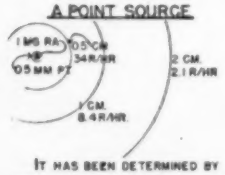
THE UNIT OF RADIUM DOSAGE - THE ROENTGEN

THE ROENTGEN IS THAT AMOUNT OF X- OR GAMMA RADIATION SUCH THAT THE ASSOCIATED CORPUSCULAR EMISSION PER 0.001293 GRAMS OF AIR PRODUCES, IN AIR, IONS CARRYING ONE ELECTROSTATIC UNIT OF QUANTITY OF ELECTRICITY OF EITHER SIGN.

0.001293 GRAMS IS THE MASS OF ONE CUBIC CENTIMETER OF AIR UNDER STANDARD CONDITIONS. THE ENERGY OF THE ASSOCIATED CORPUSCULAR RADIATION - OR SECONDARY BETA RADIATION - MUST BE COMPLETELY USED IN PRODUCING IONIZATION, AND NO OTHER RADIATION MAY CONTRIBUTE TO THIS.

A DEFINITE QUANTITY OF RADIUM UNDER A DEFINITE SET OF PHYSICAL CONDITIONS ALWAYS EMITS THE SAME AMOUNT OF RADIATION, HENCE IT IS NOT NECESSARY TO CALIBRATE RADIUM SOURCES AS IT IS X-RAY TUBES. WHEN THE OUTPUT OF A SOURCE HAS ONCE BEEN DETERMINED IT WILL BE KNOWN TO BE THE SAME FOR MANY YEARS UNLESS IT IS DAMAGED. DOSES DELIVERED BY A VARIETY OF DEFINITE APPLICATORS CAN BE CALCULATED FROM DATA OBTAINED BY PHYSICAL MEASUREMENTS AND TABULATED ONCE FOR ALL.

A POINT SOURCE



IT HAS BEEN DETERMINED BY PHYSICAL MEASUREMENTS THAT A POINT SOURCE OF ONE MILLIGRAM OF RADIUM, SURROUNDED BY ONE-HALF MILLIMETER OF PLATINUM, DELIVERS 8.4 ROENTGENS PER HOUR AT A DISTANCE OF ONE CENTIMETER. FOR OTHER DISTANCES THE NUMBER OF ROENTGENS DELIVERED VARIES IN ACCORDANCE WITH THE INVERSE SQUARE LAW.

A TUBULAR SOURCE

WHEN ONE MILLIGRAM OF RADIUM IS UNIFORMLY DISTRIBUTED ALONG A TUBE, THE NUMBER OF ROENTGENS DELIVERED OPPOSITE ITS CENTER IS LESS THAN THAT AT THE SAME DISTANCE FROM A POINT SOURCE OF THE SAME STRENGTH AND FILTRATION. TWO FACTORS ARE RESPONSIBLE FOR THIS - A) MOST OF THE RADIUM IS FARTHER AWAY FROM THE POINT IN QUESTION. B) THE RADIATION FROM THE OUT-LYING RADIUM PASSES OBLIQUELY THROUGH THE FILTER AND HENCE IS ACTUALLY FILTERED MORE STRONGLY.

THE DOSE UNDER THE END OF THE TUBE IS LESS THAN THAT UNDER ITS CENTER FOR THE SAME REASONS. DOSES AT DIFFERENT DISTANCES DO NOT FOLLOW THE INVERSE SQUARE LAW, BECAUSE THE DISTANCES FROM THE OUT-LYING REGIONS DO NOT VARY IN THE SAME MANNER AS FROM THE CENTER, AND ALSO BECAUSE THE OBLIQUE FILTRATION IS DIFFERENT AT DIFFERENT DISTANCES. THE LONGER THE TUBE THE GREATER THE DIFFERENCE BETWEEN DOSES UNDER CENTER AND END, THE GREATER THE VARIATION FROM THE POINT SOURCE VALUE, AND THE GREATER THE DIVERGENCE FROM THE INVERSE SQUARE LAW.

APPLICATORS

FOR ALL APPLICATORS THE SAME CONSIDERATIONS APPLY AS FOR TUBES. DOSE AT ANY POINT DEPENDS ON AREA AND DISTANCE OF APPLICATOR AND MANNER IN WHICH RADIUM SOURCES ARE DISTRIBUTED IN IT. IF TUBES ARE UNIFORMLY SPACED OVER SURFACE OF A PLAQUE OR MOULD THE DOSE IS GREATEST UNDER CENTER. IT IS DESIRABLE TO HAVE FAIRLY UNIFORM DISTRIBUTION OF DOSE OVER IRRADIATED AREA; THIS CAN BE ACCOMPLISHED BY CONCENTRATING MOST OF THE SOURCES TOWARD PERIPHERY OF APPLICATOR. PATERSON AND PARKER HAVE WORKED OUT SIMPLE RULES FOR SUCH DISTRIBUTIONS. (SEE LATER CHARTS AND BRITISH JOURNAL OF RADIOLOGY, VOL. VII, PAGE 592, OCT. 1934) FOR SMALL APPLICATORS AND SHORT DISTANCES ALL RADIUM SHOULD BE ARRANGED AROUND THE PERIPHERY. IF DIAMETER OF APPLICATOR IS MORE THAN FIVE TIMES DISTANCE, SOME RADIUM SHOULD BE PUT AT CENTER.

Comparison of Dosage from Uniform and Recommended Distributions

AREA 4 × 4 CM. NEEDLES 4 CM. ACTIVE LENGTH, 4 MG. RADIUM CONTENT EACH

UNIFORM

AROUND PERIPHERY

ROENTGENS PER HOUR ON SKIN ONE CM. BELOW INDICATED POINTS

A- 63	C- 39	A'- 37	C'- 42
B- 48	D- 31	B'- 42	D'- 37
MAXIMUM VARIATION 100%		MAXIMUM VARIATION 14%	

EFFECTS OF DIFFERENCES IN FILTRATION

FOR MOST PUBLISHED CHARTS AND TABLES OF RADIUM DOSAGE, AND FOR ALL THOSE IN THIS EXHIBIT, THE RADIUM IS ASSUMED TO BE FILTERED BY 0.5 MM. PLATINUM OR ITS EQUIVALENT. FOR LIGHTER OR HEAVIER FILTRATION, VALUES OBTAINED FROM THESE CHARTS CAN BE CORRECTED IN ACCORDANCE WITH TABLE BELOW. INCREASE IN FILTER RESULTS IN DECREASE OF OUTPUT IN ROENTGENS, AND IN INCREASE IN NUMBER OF MILLIGRAM-HOURS NEEDED TO DELIVER A SPECIFIED NUMBER OF ROENTGENS.

CORRECTION FACTORS FOR VARIOUS FILTERS

METAL	THICKNESS IN MM.	NO. OF ROENTGENS TRANSMITTED RELATIVE TO NO. TRANSMITTED BY 0.5 MM. PT.	NO. OF MG-HR REQUIRED TO DELIVER A SPECIFIED NO. OF ROENTGENS, RELATIVE TO NO. FOR 0.5 MM. PT
PLATINUM (OR GOLD)	0.3	111	90
	0.5	100	100
	1.0	88	113
	1.5	82	122
	2.0	78	128
	3.0	73	137
LEAD	0.5	118	85
	1.0	97	103
	2.0	88	113
	3.0	83	121
SILVER	0.5	128	78
	1.0	97	103
	2.0	94	107
	3.0	90	111
BRASS (OR COPPER OR MONEL OR STEEL)	0.5	160	62
	1.0	111	90
	2.0	100	100
	3.0	95	105
	4.0	92	109

QUIMBY TABLE FOR SURFACE APPLICATORS

NUMBER OF ROENTGENS PER 100 MG-HR DELIVERED TO CENTER OF FIELD AT VARIOUS DISTANCES FROM FLAT APPLICATORS OF VARIOUS SIZES AND SHAPES. RADIUM UNIFORMLY DISTRIBUTED OVER SURFACE. FILTER EQUIVALENT TO 0.5 MM. PT.

DISTANCE CM	CIRCULAR APPLICATORS DIAMETER - CM					
	1	2	3	4	5	6
0.5	2300	1340	968	587	456	333
1.0	740	575	453	334	270	212
1.5	354	309	250	210	178	147
2.0	202	185	165	143	126	109
2.5	136	126	115	105	93	79
3.0	92	87	82	76	70	64
	SQUARE APPLICATORS LENGTH OF SIDE - CM					
	1	2	3	4	5	6
0.5	2150	1260	870	510	400	308
1.0	710	534	420	306	248	196
1.5	340	290	242	196	167	136
2.0	200	176	155	134	117	100
2.5	137	126	112	99	88	73
3.0	92	87	79	72	66	60
	RECTANGULAR APPLICATORS DIMENSIONS - CM					
	1 × 15	2 × 3	3 × 4	4 × 6	6 × 9	8 × 12
0.5	1960	970	700	350	176	105
1.0	680	466	365	235	138	91
1.5	337	270	215	161	106	74
2.0	198	170	140	115	81	60
2.5	139	119	101	88	56	50
3.0	99	84	75	66	52	40

Example of Use of Table

PLAQUE IS MADE UP OF BRASS BOX WITH RADIUM TUBES UNIFORMLY SPACED OVER SURFACE. ACTIVE AREA 4 CM. SQUARE, TOTAL RADIUM CONTENT 60 MG. RADIUM TUBES FILTERED BY 0.5 MM. PT. BRASS BOTTOM OF BOX 1 MM. THICK, RADIUM-SKIN DISTANCE IS 2 CM.

A. HOW MANY ROENTGENS ARE DELIVERED TO THE SKIN IN AN APPLICATION OF 3 HOURS?

60 MG FOR 3 HOURS = 180 MG-HR. FROM TABLE, A SQUARE APPLICATOR 4 CM ON A SIDE, FILTERED BY 0.5 MM PT. DELIVERS 134 R PER 100 MG-HR AT 2 CM. FOR 180 MG-HR DOSE IS 1.8 × 134 = 231 R. CORRECTION FOR ADDITIONAL BRASS REDUCES THIS TO 95%. 0.95 × 231 = 220 R DOSE ON SKIN FOR SPECIFIED TREATMENT.

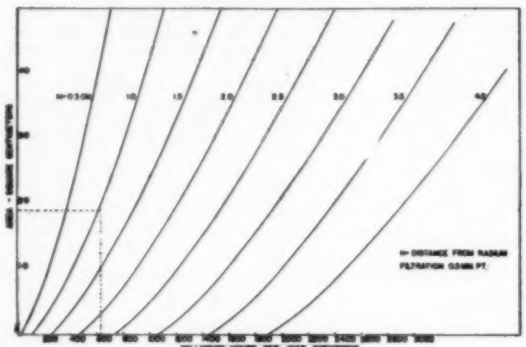
B. HOW MANY ROENTGENS ARE DELIVERED 1 CM. BELOW SKIN IN SAME TREATMENT?

ONE CM. BELOW SKIN GIVES RADIUM-TISSUE DISTANCE OF 3 CM. THIS APPLICATOR GIVES 72 R PER 100 MG-HR AT THIS DISTANCE. TISSUE ABSORPTION AND SCATTER MAY BE NEGLECTED. CORRECTION FOR BRASS MUST BE APPLIED. 72 × 1.8 × 0.95 = 122 R, DOSE AT 1 CM. DEPTH.

C. HOW LONG MUST APPLICATOR BE LEFT IN PLACE TO DELIVER 1000 R AT 1 CM. DEPTH?

100 MG-HR, CORRECTED FOR EXTRA BRASS, DELIVERS 72 × 0.95 = 68 R AT 1 CM. DEPTH. TO DELIVER 1000 R WILL REQUIRE $\frac{1000}{68} \times 100 = 1460$ MG-HR. SINCE 60 MG ARE IN APPLICATOR, TIME WILL BE $\frac{1460}{60} = 24.3$ HOURS.

PATERSON AND PARKER CHART FOR SURFACE APPLICATORS



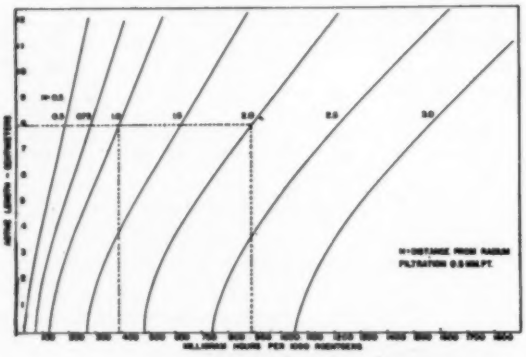
RULES FOR DISTRIBUTION OF RADIUM

TREAT AREA SLIGHTLY LARGER THAN LESION
USE CIRCLES WHENEVER POSSIBLE
AT LEAST 6 CONTAINERS TO MAKE CIRCLE
SPACING BETWEEN ENDS OF TUBES NOT GREATER THAN DISTANCE H
FOR DIAMETER LESS THAN 3H, ONE CIRCLE
DIAMETER 3-6H, ADD CENTRAL SPOT
LARGER DIAMETERS, CONCENTRIC CIRCLES
IF SQUARE, RECTANGLE, OR IRREGULAR AREA IS USED, FOLLOW SAME GENERAL PRINCIPLES
IF SIDE OF SQUARE IS MORE THAN 2H, USE LINES OF RADIUM TO DIVIDE IT INTO STRIPS OF WIDTH 2H
FOR RECTANGLES WITH RATIO OF SIDES 3 OR 4 TO 1, INCREASE MG-HR BY 10 %

EXAMPLE (GIVEN BY PATERSON AND PARKER)

EPITHELIOMA OF DORSUM OF HAND. LESION 3.5 CM. DIAM. TREAT AREA 5 CM DIAM. = 19 SQ CM.
TO GIVE 6000 R TO SURFACE OF LESION USING WAX MOULD SUCH THAT H=1 CM.
FOR THIS FIELD AND DISTANCE 1000 R REQUIRE 630 MG HR. 6000 R REQUIRE 3780 MG HR.
AVAILABLE TUBES 2 MG EACH, ACTIVE LENGTH 1.5 CM., FILTER 0.5 MM. PT.
USE CIRCLE OF 6 TUBES AND 1 FOR CENTER SPOT, TOTAL 14 MG.
IRRADIATION TIME $\frac{3780}{14}$ = 270 HOURS. 10 HOURS DAILY FOR 27 DAYS MIGHT BE CONVENIENT
DEPTH DOSE AT BASE OF LESION 0.5 CM DEEP: H=1.5, 880 MG. HR FOR 1000 R
FOR 3780 MG. HR., DOSE AT H=1.5 IS $\frac{3780}{880}$ X 1000 = 4,300 R

PATERSON AND PARKER CHART FOR LINEAR SOURCES



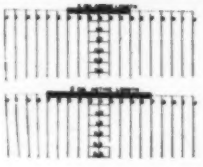
EXAMPLE (GIVEN BY PATERSON AND PARKER)

CARCINOMA OF RECTUM, ANNULAR LESION ABOUT 3 CM LONG, TO BE TREATED BY TODD'S APPLICATOR (AN INFLATABLE DUMBELL-SHAPED APPLICATOR THE DIAMETER OF WHICH IN THE MIDDLE IS 2 CM, BUT AT THE ENDS 4 CM.) DESIRED TO GIVE 10,000 R TO SURFACE OF TUMOR IN ABOUT 5 DAYS AND TO KNOW DOSE AT NORMAL MUCOSA (UNDER ABOUT 1 CM. OF TUMOR)
USE 4 TUBES OF 10 MG EACH, GIVING AN ACTIVE LENGTH OF 8 CM. FILTRATION 1.5 MM PT.
FOR TUMOR SURFACE H=1 CM., FOR AN 8 CM TUBE WITH 0.5 MM. PT. 390 MG-HR FOR 1000 R
THE EXTRA 1 MM PT. INCREASES THIS BY 22 %, TO 475 MG-HR. 10,000 R REQUIRE 4750 MG-HR.
WITH 40 MG, THIS MEANS AN EXPOSURE OF $\frac{4750}{40}$ = 119 HOURS. FOR MUCOSA AT BASE OF TUMOR, H=2 CM. 1000 R FOR 0.5 MM PT. REQUIRE 890 MG-HR. WITH ADDITIONAL FILTER, 1.085.
SINCE THE EXPOSURE DECIDED ON IS 4750 MG-HR, MUCOSA WILL RECEIVE $\frac{4750}{1.085}$ = 4400 R.

GAMMA ROENTGENS PER 100 MG-HR. DELIVERED AT VARIOUS DISTANCES FROM VARIOUS POINTS ALONG LINEAR RADIUM SOURCES (FILTER 0.5 MM. PT.)

CM. FROM TUBE	DISTANCE ALONG TUBE AXIS: CM. FROM CENTER								
	A	B	C	D	E	F	G	H	I
	CENTER	0.5	1.0	1.5	2.0	2.5	3.0	3.5	4.0
TUBE 2.0 CM. LONG									
0.5	1800	1720	910	385	195	115	76	49	33
0.75	1111	935	625	333	189	115	77	55	40
1.0	625	570	435	270	168	110	75	55	42
1.5	333	303	257	189	135	97	70	54	43
2.0	196	185	159	131	102	78	61	48	39
2.5	125	121	111	96	80	65	52	43	35
3.0	89	88	81	72	62	52	44	37	31
4.0	51	50	48	45	40	36	32	28	25
5.0	33	33	32	30	28	26	24	22	20
TUBE 3.0 CM. LONG									
0.5	1430	1333	1111	645	310	145	88	59	41
0.75	910	835	690	435	250	141	89	61	43
1.0	540	526	454	323	202	133	86	61	44
1.5	286	286	250	193	145	107	77	54	43
2.0	179	173	156	132	106	83	65	51	40
2.5	119	115	107	95	81	67	53	44	36
3.0	85	84	78	71	62	54	45	38	32
4.0	50	49	48	44	40	36	32	28	25
5.0	32	32	31	30	28	26	24	22	20

CM. FROM TUBE	DISTANCE ALONG TUBE AXIS: CM. FROM CENTER								
	A	B	C	D	E	F	G	H	I
	CENTER	0.5	1.0	1.5	2.0	2.5	3.0	3.5	4.0
TUBE 4.0 CM. LONG									
0.5	1111	1050	1000	910	500	222	118	72	48
0.75	690	667	625	453	344	192	114	73	50
1.0	435	435	418	333	256	164	107	71	50
1.5	256	238	228	193	159	120	89	62	46
2.0	161	158	148	132	111	89	70	55	44
2.5	111	107	104	92	82	71	58	48	38
3.0	82	80	78	70	62	56	47	40	33
4.0	48	48	46	44	40	36	32	29	25
5.0	32	31	30	29	28	26	24	22	20
TUBE 5.0 CM. LONG									
0.5	833	827	820	800	715	400	200	100	62
0.75	572	555	525	465	377	273	155	95	62
1.0	370	370	364	333	286	213	136	88	61
1.5	228	217	213	194	167	135	100	73	56
2.0	148	143	137	128	112	94	76	64	48
2.5	103	100	97	91	82	73	60	50	41
3.0	77	75	72	68	62	56	48	41	35
4.0	46	46	44	42	40	36	33	29	25
5.0	31	30	30	29	27	25	24	22	20
TUBE 6.0 CM. LONG									
0.5	740	735	730	715	690	605	345	187	98
0.75	500	495	490	450	418	371	244	139	84
1.0	333	333	321	310	294	250	179	115	77
1.5	200	200	196	194	175	152	118	91	67
2.0	134	132	128	123	112	99	82	66	52
2.5	96	93	91	89	83	74	62	53	43
3.0	75	72	69	66	63	57	50	42	36
4.0	44	44	42	41	39	36	33	30	26
5.0	30	30	29	28	27	25	24	22	20



EXAMPLES FOR QUIMBY TABLE OF DOSAGE FROM LINEAR SOURCES

CARCINOMA OF THE CERVIX, TO BE TREATED WITH TANDEM OF TWO CAPSULES. THE UPPER ONE CONTAINS 25 MG. RADIUM AND IS 2 CM. IN ACTIVE LENGTH, THE LOWER CONTAINS 50 MG. AND IS 3 CM. EACH IS FILTERED BY 0.5 MM. PT. BETWEEN THE ACTIVE PARTS IS A SPACE OF 0.5 CM. IT IS PLANNED TO LEAVE THE TANDEM IN PLACE 40 HR. FOR A TOTAL EXPOSURE OF 3000 MG-HR, OF WHICH THE UPPER TUBE FURNISHES 1000 AND THE LOWER 2000 MG-HR. IT IS DESIRED TO KNOW THE DOSE AT POINT P, THE LIMIT OF THE LESION AS FELT, AND AT POINT X, 4 CM. OUT FROM THE CENTER OF THE TANDEM.

POINT P 2.5 CM. OUT ON LINE C FROM LOWER TUBE = 107 R PER 100 MG-HR
2.5 CM. OUT ON LINE I FROM UPPER TUBE = 35 R PER 100 MG-HR
DOSE FROM LOWER TUBE 20 × 107 = 2140 R
FROM UPPER TUBE 10 × 35 = 350 R
TOTAL 2490 R

POINT X 4 CM. OUT ON LINE D FOR EACH TUBE. FROM LOWER = 44 R PER 100 MG-HR
FROM UPPER = 45 R PER 100 MG-HR
44 × 20 + 45 × 10 = 1330 R

CARCINOMA OF CERVIX: PARAMETRIAL NEEDLES INSERTED AS FAR LATERALLY AS POSSIBLE, LYING APPROXIMATELY IN AN ANTERO-POSTERIOR PLANE. EACH NEEDLE IS 3 CM. IN ACTIVE LENGTH AND CONTAINS 3 MG. OF RADIUM. THE FILTRATION IS 0.5 MM. PT. THE LOWER ENDS OF THE NEEDLES ARE 0.5 CM. APART, UPPER ENDS 1.5 CM. APART. THEY ARE TO BE LEFT IN PLACE 100 HOURS, HENCE EACH NEEDLE SUPPLIES 300 MG-HR. WHAT DOSES ARE DELIVERED AT POINT P, MIDWAY BETWEEN THE UPPER ENDS OF TWO NEEDLES, AND AT POINT X, 1 CM. ABOVE THE CENTER OF THE CENTER NEEDLE?

POINT P: DISTANCE 0.75 CM. ALONG LINE D FROM TWO NEAREST NEEDLES, 2 CM. ALONG LINE G FOR FARTHER ONE.

AT 0.75 CM. ON LINE D FROM 3 CM. NEEDLE = 435 R PER 100 MG-HR
AT 2.0 CM. ON LINE G = 156 R PER 100 MG-HR
2 × 3 × 435 + 3 × 156 = 3078 R

POINT X: ON LINE A FOR ALL NEEDLES DISTANCE 1 CM. FROM CENTER NEEDLE AND 1.4 CM. FROM TWO OUTSIDE ONES.

AT 1 CM. ON LINE A FROM 3 CM. NEEDLE 540 R PER 100 MG-HR
AT 1.4 CM. 317 R PER 100 MG-HR
3 × 540 + 2 × 3 × 317 = 3522 R

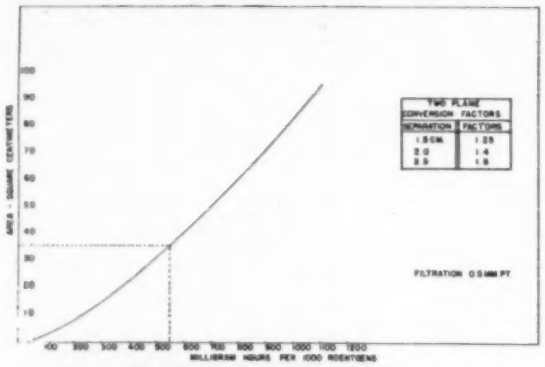
CARCINOMA OF RECTUM, ANNULAR LESION ABOUT 3 CM. LONG, PROBABLY 1 CM. THICK, TO BE TREATED BY LINEAR APPLICATOR CONSISTING OF BRASS SHELL 2 CM. IN DIAMETER, WALL THICKNESS 1 MM., CARRYING THE RADIUM TUBES IN ITS AXIS. ACTIVE LENGTH 6 CM. FILTER ON RADIUM TUBES 1.0 MM. PT. TOTAL RADIUM 75 MG. TO DELIVER 4000 R TO BASE OF TUMOR (POINT X). AT 2 CM. ON LINE A FROM 6 CM. TUBE, 134 R PER 100 MG-HR WITH 0.5 MM. PT. ADDITIONAL PT. AND BRASS REDUCE THIS TO 85 %, OR 112 R.

$\frac{4000}{112} \times 3750$ MG-HR. FOR THIS DOSE FOR 75 MG., THE EXPOSURE IS $\frac{3750}{75} = 475$ HR.

DOSE TO SURFACE OF TUMOR IS OF INTEREST (POINT Y). AT 1 CM. ON LINE A FROM 6 CM. TUBE, 353 R PER 100 MG-HR, 280 R WITH FILTER CORRECTION 35.7 × 280 = 10,000 R.

DOSE AT UPPER EDGE OF TUMOR IS ALSO IMPORTANT. ASSUME IT EXTENDS UPWARD 2 CM. AND IS 0.5 CM. THICK AT EDGE. (POINT Z) AT 1.5 CM. ON LINE E FROM 6 CM. TUBE, 175 R PER 100 MG-HR. 147 R WITH FILTER CORRECTION 35.7 × 147 = 5250 R.

PATERSON AND PARKER CHART FOR PLANE IMPLANTS



EXAMPLE (GIVEN BY PATERSON AND PARKER)

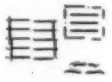
TUMOR OF SKIN - TWO PLANE IMPLANT. LESION IS A FLAT SOLID IMPLANTATION IN ABDOMINAL WALL FOLLOWING EXCISION OF INTRA-ABDOMINAL TUMOR. DIMENSIONS 5×6×1.5 CM. TO GIVE 6000 R BY 2-PLANE IMPLANT, ONE LARGER PLANE UNDER-CUTTING MASS, ONE DIRECTLY THROUGH IT, PLANES 1.5 CM. APART. LOWER 7×6 = 42 SQ. CM. UPPER 5×6 = 30 SQ. CM. AVERAGE 36 SQ. CM. FOR THIS AREA 550 MG-HR PER 1000 R. FACTOR FOR 2-PLANE SEPARATION, 25 %, INCREASES THIS TO 690 MG-HR PER 1000 R. FOR 6000 R, EXPOSURE WILL BE 6×690 = 4140 MG-HR. USE WEAK NEEDLES TO STAY IN PLACE ABOUT 7 DAYS. $\frac{4140}{24 \times 7}$ = 25 MG RA. THIS SHOULD BE DIVIDED IN RATIO OF AREAS, UPPER PLANE 10 MG., LOWER, 15 MG. SINCE EACH IS LARGER THAN 25 SQ CM., PLACE HALF OF RADIUM AROUND PERIPHERY AND DISTRIBUTE HALF THROUGH AREA. DIAGRAM SHOWS SUITABLE ARRANGEMENT FOR NEEDLES LOADED WITH 0.33 MG. PER CM.

RULES FOR DISTRIBUTION OF RADIUM

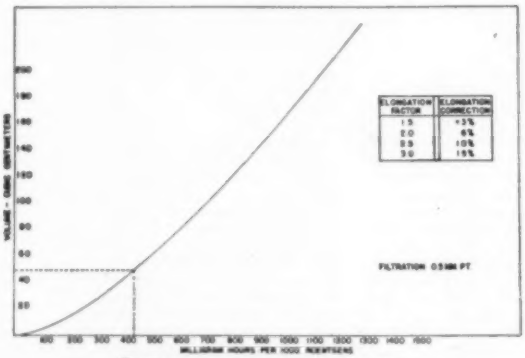
FOR A SINGLE PLANE, HAVING AN AREA LESS THAN 25 SQ CM ALL RADIUM SHOULD BE PLACED AROUND PERIPHERY, AREAS LARGER THAN THIS SHOULD HAVE HALF AROUND PERIPHERY AND HALF UNIFORMLY DISTRIBUTED THROUGH THE LESION.

NEEDLES IN PARALLEL PLANES SHOULD HAVE THE ENDS CROSSED BY OTHER NEEDLES AT RIGHT ANGLES. IF THIS CANNOT BE DONE, THE IRRADIATED AREA MUST BE CONSIDERED 10 % LESS FOR EACH UNCROSSED END. PARALLEL NEEDLES SHOULD NOT BE MORE THAN 1 CM APART OR FROM CROSSING ENDS.

FOR TWO PLANES, THE RADIUM SHOULD BE ARRANGED IN THE SAME MANNER AND THE PLANES SHOULD BE PARALLEL. IF THE PLANES DIFFER IN AREA, THE AREA TO BE USED FOR THE GRAPH IS THE AVERAGE OF THE TWO, AND THE TOTAL RADIUM DIVIDED IN PROPORTION TO THE AREAS.



PATERSON AND PARKER CHART FOR VOLUME DOSAGE



EXAMPLE (GIVEN BY PATERSON AND PARKER)

EPITHELIOMA OF FLOOR OF MOUTH - VOLUME IMPLANT. CENTER OF LESION IS IN REGION OF FRENUM OF TONGUE, GROWTH EXTENDS FORWARD INTO FLOOR AND UPWARD INTO SUBSTANCE OF TONGUE. WITH TONGUE STITCHED DOWN TO FLOOR OF MOUTH THIS TYPE OF LESION IS WELL TREATED AS A VERTICAL CYLINDER, WITH NEEDLES IN LIP AS ANTERIOR PORTION OF BELT, AND BOTH ENDS CROSSED, UPPER END IN DORSUM OF TONGUE, LOWER END SUBMENTAL. PROPOSED DOSE 6000 R. VOLUME OF ELLIPTIC CYLINDER 4.5×4 CM., 3.5 CM HIGH = 49.4 CU CM. FOR 1000 R USE 450 MG-HR. NO CORRECTION FOR OPEN ENDS OR ELONGATION. FOR 6000 R = 6×450 = 2700 MG-HR. IF RADIUM IS TO BE LEFT IN PLACE ONE WEEK, THIS MEANS $\frac{2700}{24 \times 7}$ = 16 MG. THIS SHOULD BE DIVIDED 8 MG IN BELT, 4 MG IN CORE, 2 MG IN EACH END. VARIOUS COMBINATIONS OF WEAK NEEDLES CAN BE USED.

RULES FOR DISTRIBUTION OF RADIUM

ANY VOLUME SHOULD BE VISUALIZED AS CONSISTING OF TWO COMPONENTS, THE OUTER SURFACE OR "RIND", AND THE "CORE" OR ENTIRE VOLUME BEING IRRADIATED. THE TOTAL RADIUM SHOULD BE DIVIDED AS FOLLOWS FOR THE VARIOUS SHAPES -

SPHERE - SHELL, THREE-FOURTHS: CORE, ONE-FOURTH.

CYLINDER - BELT, ONE-HALF: EACH END, ONE-EIGHTH: CORE, ONE-FOURTH.

CUBOID - EACH SIDE, ONE-EIGHTH: EACH END, ONE-EIGHTH: CORE, ONE-FOURTH.

THE RADIUM ON THE SURFACE IS TO BE EVENLY SPACED. THAT FOR THE CORE IS TO BE EVENLY DISTRIBUTED THROUGHOUT THE VOLUME, NOT ALL AT THE CENTER.

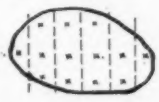
QUANTITIES OF INTERSTITIAL RADIATION NECESSARY TO DELIVER 1000 ROENTGENS IN VARIOUS VOLUMES FILTER 0.5 MM PT (QUIMBY)

DIAMETER OF SPHERICAL MASS – CENTIMETERS										
1	1.5	2.0	2.5	3.0	3.5	4.0	4.5	5.0	5.5	6.0
VOLUME OF MASS – CUBIC CENTIMETERS										
0.5	2	4	8	15	25	35	50	65	90	115
MILLIGRAM - OR MILLICURIE-HOURS TO DELIVER 1000 ROENTGENS										
35	80	135	250	330	420	540	680	830	1000	1160
MILLICURIES DESTROYED TO DELIVER 1000 ROENTGENS										
0.25	0.6	1.0	1.9	2.5	3.2	4.0	5.0	6.2	7.5	8.7

EXAMPLES

A NECK NODE SECONDARY TO CARCINOMA OF TONGUE, 2 × 3 × 3 CM, TO BE IMPLANTED WITH GOLD SEEDS TO GIVE 7000 R. FROM TABLE FOR 3 CM. SPHERE, 1000 R. REQUIRE 2.5 MC. DESTROYED, 7 × 2.5 = 17.5 MC. A GOOD DIVISION WOULD BE 12 SEEDS OF APPROXIMATELY 1.5 MC. EACH.

A SOFT PART TUMOR 3 × 4 × 7 CM. IS TO BE IMPLANTED WITH NEEDLES FOR A DOSE OF 4000 R. AVAILABLE NEEDLES ARE 3 CM. ACTIVE LENGTH, 1 MG. EACH AND 4 CM. ACTIVE LENGTH, 2 MG. EACH, FILTER 0.5 MM PT. 3 × 4 × 7 = 84 CU. CM. INTERPOLATING BETWEEN VALUES FOR 65 AND 90 CU. CM., TO GIVE 1000 R. IN 84 CM. REQUIRES 960 MG-HR. 4 × 960 = 3840 MG-HR. ONE SATISFACTORY SCHEME. ARRANGE 13, 3 CM. NEEDLES IN 3 PLANES 1.5 CM. APART, AS SHOWN BY CROSSES. CROSS ENDS OF THESE ABOVE AND BELOW WITH 4 CM. NEEDLES - DOTTED LINES. TOTAL RADIUM - (3 × 1 + 10 × 2 = 33 MG. $\frac{3840}{33}$ = 116 HR. - 4 DAYS 20 HR.



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EDITORIAL

ROENTGEN KYMOGRAPHY AND ROENTGEN ELECTROKYMOGRAPHY

ON JULY 7, 1911, Sabat[1] presented before the Medical Society of Lwow a procedure for the roentgen demonstration of the movements of the diaphragm, heart and aorta. This was based on the use of a simple apparatus, consisting of a single slit diaphragm, which was placed against a selected part of the organ in motion and through it a roentgenographic recording was made while the plate was moving perpendicular to the slit. The image appeared in the form of waves, the peaks of which depended on the amplitude of the movements of the organ in question. In the following year Gött and Rosenthal[2] coined for the procedure the name roentgen kymography.

Although a more accurate analysis of the movements of inner organs than could be seen roentgenoscopically constituted a desired goal from the very incipiency of the science of roentgenology, these initial studies of Sabat, Gött and Rosenthal, as well as those of Crane,[3] and a few others, were soon followed by a rather long period of inactivity. The main reason no doubt must be sought in the technical limitations existing at that. It was not until 1928 that the method was revived. Stumpf[4] in this year introduced the grid with multiple parallel slits replacing the diaphragm with one or a few slits used by the earlier investigators. Since then the progress has been more swift. Numerous studies have been carried out and several types of apparatus have been constructed aiming to meet various clinical requirements.

For obvious reasons, the greatest achievement was made in conjunction with the examination of the movements of the heart. The apparatus used for this purpose is mostly of two types. In the one type, the grid which is placed between the thorax and the film moves perpendicular to the slits during the entire exposure; in the other, the grid is stationary and the film is moved till fully exposed. By allowing for wider slit spacing and by changing the speed of either grid or film movement it is possible to obtain a varying spread of the waves, leading in certain instances to rather characteristic forms. Some investigators prefer to use many short slits with adjustable spacing and a very fast speed to gain the maximum of wave spread possible. Hirsch,[5] after an exhaustive study, devised a system which permits a synchronization of the kymographic recording with the electrocardiographic tracing as well as a simultaneous registration of the kymogram and of the heart sounds. This latter he called kymophonoroentgenography.

[1] Sabat, B. On a procedure of roentgenographic demonstration of the movements of the diaphragm, heart and aorta. (In Polish.) *Lwowski tygodnik lekarski*, 1911, *6*, July 13, No. 28; also, Sabat, B. Ueber ein Verfahren der röntgenographischen Darstellung der Bewegungen innerer Organe (des Herzens, der Aorta, des Zwerchfells). *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1913, *20*, 42–44.

[2] Gött, T., and Rosenthal, J. Ueber ein Verfahren zur Darstellung der Herzbewegung mittels Röntgenstrahlen (Röntgenkymographie). *München. med. Wchnschr.*, 1912, *59*, 2033–2035. Translated: Gocht (name misspelled), T., and Rosenthal, J. Roentgenkymography; a roentgenographic method of demonstrating the movement of the heart. *Arch. Roentg. Ray*, 1912–1913, *17*, 378–384.

[3] Crane, A. W., Roentgenocardiograms: polygraphic strip tracings of heart chambers by the roentgen ray. Am. J. Roentgenol., 1916, *3*, 374–375; also, Roentgenology of the heart. *Ibid.*, 513–524.

[4] Stumpf, P. Die Gestaltänderung des schlagenden Herzens im Röntgenbild. *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1928, *38*, 1055–1067.

[5] Hirsch, I. S. The recording of cardiac movements and sounds by the roentgen ray (kymophonoroentgenography). *Radiology*, 1934, *22*, 403–422; also, Hirsch, I. S. The application of kymoroentgenography to the diagnosis of heart disease. *Radiology*, 1934, *23*, 720–737.

It may be said that at the present time the kymographic method has a definite practical value in diagnosing certain morphologic and structural abnormalities of the heart. It also permits a more accurate estimation of the configuration of the heart as a whole or of any of its chambers, both in systole and diastole. Friedman and Friedman[6] furthermore are of the opinion that the character of the waves, the time relationship of the various pulsations and the amplitude of the pulsations may be used in the presence of cardiac or extracardiac disorders to express the functional capacity of the heart and to prognosticate the response to therapeusis.

While it is true that cardiokymography has contributed notably to the more accurate diagnosis of certain clinical entities, it is felt that, as a whole, many of the changes observed are not as yet well understood. In particular, the interpretation of the forms and amplitudes of the various types of waves represents considerable difficulty. The spreading out of the waves by increasing the speed of the moving grid or film has helped, but since the reading of the kymographic record is visual, some of the finer points characterizing the movements are missed regardless of how well spread the wave may be.

Since 1942 Morgan[7] has published a series of excellent articles dealing with the development of a very sensitive instrument capable of automatically controlling the time of exposure in roentgenography under various conditions. This instrument consists essentially of a combination of multiplier phototube and a small fluorescent screen activating it, a vacuum tube amplifier and an automatic timing mechanism. The roentgen beam after passing through the patient exposes the roentgen film and at the same time acts on the phototube-fluorescent screen assembly putting in operation the automatic timer. Since the magnitude of the small current flowing through the phototube is proportional to the intensity of the roentgen beam reaching it after it has traversed the body, the relay of the timer terminates the exposure as soon as a predetermined level is reached. The manifold applicability of the instrument is now generally recognized and the practical methods making use of it are increasing daily.

It had occurred to Chamberlain that it might be worth while to introduce a similar arrangement for the recording of the movements of the heart. This author had already been engaged since 1926[8] in a study of the possibility of graphic registration of the cardiac motion by means of superimposed serial roentgenograms obtained with the aid of the Ruggles roentgen cinematograph. By marking the change of position of selected points of the heart silhouette in chronologic order he obtained curves which were somewhat more accurate than the ordinary kymographic curves. With the advent of Morgan's discovery of the phototimer Chamberlain suggested to Henny and Boone that a photoelectric pick-up of the movements of the heart might be advantageously combined with an electrocardiographic type of registration. The result was an apparatus producing a large beam type tracing of a roentgenoscopically preselected point of the heart border on bromide paper for as many pulsations as necessary.

In a first publication Henny and Boone[9] gave a detailed description of this new apparatus, which is called roentgen electrokymograph. The roentgen-ray pick-up device consists of an RCA 931-A multiplier phototube and a small fluorescent screen

[6] Friedman, L. J., and Friedman, P. S. Practical cardiokymography; its significance in evaluating cardiac function. AM. J. ROENTGENOL. & RAD. THERAPY, 1944, *51*, 29–32.

[7] Morgan, R. H. Exposure meter for roentgenography. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *47*, 777–784; also, Studies in roentgenographic exposure meter design. *Ibid.*, 1942, *48*, 88–98; also, A photoelectric timing mechanism for the automatic control of roentgenographic exposure. *Ibid.*, 1942, *48*, 220–228; also, The control of diagnostic quality in roentgenograms of the chest. *Ibid.*, 1943, *50*, 149–161.

[8] Chamberlain, W. E., and Dock, W. Motion of the heart in disease of the mitral valve; cinematographic roentgen-ray studies *Arch. Int. Med.*, 1927, *40*, 521–531.

[9] Henny, G. C., and Boone, B. R. Electrokymograph for recording heart motion utilizing the roentgenoscope. AM. J. ROENTGENOL. & RAD. THERAPY, 1945, *54*, 217–229.

mounted in a light-tight metal housing which also supports a lead diaphragm. The purpose of the lead diaphragm is to limit the roentgen beam reaching the phototube to a narrow rectangular area which with the aid of a rotating pulley is placed with the longest dimension at right angles to the part of the heart silhouette to be examined. The entire pick-up unit is fastened with clamps on the patient's side of the regular fluoroscope. When properly centered the pulsating heart shadow moves across the rectangle, covering more of it in diastole and less of it in systole. This alternation produces a variation of the average intensity of the roentgen rays over the whole rectangular area with the result that the fluorescent screen activates the phototube in varying intensities. Thus the motion of the particular portion of the silhouette border to be examined is translated into a correspondingly varying electrical current. The graphic recording of this current is done by means of a standard electrocardiographic machine. In order to establish proper time relationship a simultaneous recording of the right carotid artery pulse is made with each heart motion curve. A small bakelite cup 3 cm. in diameter is placed over the right carotid artery by means of a light wooden clamp and the pulse is inscribed by means of a simple system at the bottom of the roentgen electrokymographic curve. The timing is expressed in intervals of 0.10 second.

As one would expect there were several technical difficulties which had to be overcome before the method could be recommended for routine practical use. In the April issue of this JOURNAL, Henny, Boone and Chamberlain[10] published an improved model of the roentgen electrokymograph which at the same time represents considerable simplification. This new version of the apparatus records the movements of the border of the heart and great vessels with excellent clarity and remarkable fidelity. Interfering ripples and other disturbing factors have been successfully eliminated.

As to its clinical value, it appears that roentgen electrokymography has at least three great advantages over ordinary roentgen kymography. In the first place, it produces on the record amplitudes up to 20 mm. or more whereas the amplitudes of the roentgen kymograms are only from 4 to 8 mm. high; second, it registers the wave forms in a sharp and detailed manner, permitting a more precise reading than is the case with the roentgen kymographic waves; and third, it expresses the radial motion of a point and not merely its horizontal component as does roentgen kymography.

There can be no doubt that the roentgen electrokymograph will soon prove its usefulness in the examination of many hitherto undiagnosable conditions of the cardiovascular system. As times goes on, it will also suggest itself for the investigation of the movements of other internal organs of the body.

T. LEUCUTIA

[10] Henny, G. C., Boone, B. R., and Chamberlain, W. E. Electrokymograph for recording heart motion, improved type. AM. J. ROENTGENOL. & RAD. THERAPY, 1947, *57*, 409–416.

We regret to announce the death of Prof. Dr. Med. Alban Köhler, formerly of Wiesbaden, of a heart attack on February 26, 1947, a few days before his seventy-third birthday. Dr. Köhler was well known to all roentgenologists and was an honorary member of the American Roentgen Ray Society. A more extended account of Dr. Köhler's life and work will be published in a future issue of the JOURNAL.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Haddon Hall, Atlantic City, N. J., Sept. 16–19, 1947.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: Seaside Hotel, Atlantic City, N. J., June 9–10, 1947.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1947, to be announced.

American College of Radiology
Secretary, Mac F. Cahal, 20 N. Wacker Drive, Chicago 6. Annual meeting: Haddon Hall, Atlantic City, N. J., June 8, 1947.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio. Annual meeting: Atlantic City, N. J., June 9–13, 1947.

Alabama Radiological Society
Secretary, Dr. John D. Peake, Mobile Infirmary, Mobile Ala. Next meeting time and place of Alabama State Medical Association.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. H. Levy, 1354 Carroll St., Brooklyn 13, N. Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Chicago Roentgen Society
Secretary, Dr. T. J. Wachowski, 310 Ellis Ave., Wheaton, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. George L. Sackett, 10515 Carnegie Ave. Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Denver Radiological Club
Secretary, Dr. W. C. Huyler, 1619 Milwaukee, Denver 6, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida Radiological Society
Secretary, Dr. J. M. Dell, Jr., 333 W. Main St., S., Gainesville, Fla. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. J. A. Campbell, Indiana University Hospitals, Indianapolis 7. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Louisville Radiological Society
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Louisville General Hospital.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at

* Secretaries of societies not here listed are requested to send the necessary information to the Editor.

time of Minnesota State Medical Association, the other in the fall.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. O. A. Neely, 924 Sharp Bldg., Lincoln, Neb. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. Hemphill, 323 Professional Bldg., Charlotte 2, N. C. Meets in May and October.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. P. E. Russo, 230 Osler Bldg., Oklahoma City, Okla. Three regular meetings annually.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. C. L. Stewart, Jefferson Hospital. Meets first Thursday of each month, October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. L. M. J. Freedman, 115 South Highland Ave. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

PORTLAND ROENTGEN CLUB
Secretary, Dr. Selma Hyman, University of Oregon Medical School, Portland, Oregon. Meets monthly 2d Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Robert M. Lowman, Grace-New Haven Community Hospital, New Haven 11, Conn. Meets bimonthly on second Thursday, at place selected by Secretary.

RADIOLOGICAL SECTION, LOS ANGELES CO. MED. ASSN.
Secretary, Dr. Moris Horwitz, 2009 Wilshire Blvd., Los Angeles 5, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. W. H. Seward, Orange Memorial Hospital, Orange, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September.

SAN DIEGO ROENTGEN SOCIETY
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

UTAH RADIOLOGICAL CONFERENCE
Secretary, Dr. Henry H. Lerner, School of Medicine, University of Utah, Salt Lake City 1. Meets 1st and 3rd Thursdays monthly from 7:30 to 10 P.M., Salt Lake County General Hospital, September to June.

UTAH STATE RADIOLOGICAL SOCIETY
Secretary, Dr. M. Lowry Allen, Judge Bldg., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St. Richmond, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle.

Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. Ivan J. Miller, 2000 Van Ness Ave. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Lane Hall, Stanford University Hospital and second six months at Toland Hall, University of California Hospital.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Roentgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 23 Welbeck St., London, W.1 England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 2100 Marlowe Ave., Montreal 28, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Francaise d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306 Lima, Peru. Meetings held monthly except during January, February and March, at the Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Continental Europe

Société belge de radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Alliés, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary: MUDr. Roman Blána, Praha XII, Korunní 160, Czechoslovakia.

Polish Society of Radiology
First post-war inaugural meeting will be held in Warsaw, May 22 and 23, 1947.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, Nowogrodzka 59, Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. Babaiantz, Geneva.
Secretary for German language, Dr. Max Hopf, Effingerstrasse 49, Bern. Meets annually in different cities.

SHORTAGE OF PAPER NECESSITATES USING SUBSTITUTE PAPER FOR REPRINTS

The paper situation continues to be difficult; in fact, it is more difficult to secure paper now than it was a few months ago. The George Banta Publishing Company which prints the AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY has been fortunate in being able to secure enough of the glossy enamel paper to print the regular issues of the JOURNAL. However, the scarcity of this paper has made it necessary in recent months to print the reprints of articles published in the JOURNAL on substitute paper of not such a good grade. This naturally results in much less satisfactory appearing reprints and will be a source of disappointment to those ordering reprints. The Editor, the Publisher and the Printer all regret the necessity for this substitution. It is, however, a situation which cannot be avoided at present but is one which will be rectified at the earliest possible moment.

COURSE IN APPLICATION OF NUCLEAR PHYSICS TO THE BIOLOGICAL AND MEDICAL SCIENCES

The University of California Medical School, in association with University Extension, University of California, announces a Course in the Application of Nuclear Physics to the Biological and Medical Sciences. This course will be given at the Medical Center, in San Francisco, from June 30 through July 18, 1947. It will consist of didactic lectures, laboratory demonstrations and seminars for round table discussions, and will be open to individuals in the fields of medical and biological research. Requests for detailed information are to be addressed to Stacy R. Mettier, M.D., Head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco 22, California.

BIOLOGICAL PHOTOGRAPHIC EXHIBITION

An International Exhibition of Biological Photography of interest to all photographers in the scientific field will be held September 10–27, 1947, under the sponsorship of the Biological Photographic Association in Rochester, New York. The Exhibition will be on display in the beautiful gallery of the Rundel Building of the Rochester Public Library.

The scope of the Exhibition, which will be held in conjunction with the 1947 Convention of the Biological Photographic Association, includes scientific photography of all biological subjects. The term, "biological," has been considered in its broadest sense. The major fields of medicine and natural history will constitute the majority of photographic interest. However, all aspects of biology—zoology, genetics, osteology, paleontology, etc., entymology, ornithology, ecology, and geology, as it pertains to living organisms, are subjects for exhibition. Scientific record photography is not to be ignored.

It is the desire of the Exhibition Committee to include all photographic techniques in the preparation of black-and-white and color still and motion-picture photographic material. The technical procedures desired are those of general photography, photomicrography (electron and visible light), radiography (except as applied in routine roentgenography), autoradiography, microradiography, electron diffraction of animal or plant tissues, high-speed photography, infrared, ultraviolet, polarized light records, etc.

Membership in the Biological Photographic Association is *not* a requisite for participation in the Exhibition.

Information concerning the convention program of the Biological Photographic Association to be held September 10–13, 1947, may be secured from Mr. John V. Butterfield, 635 St. Paul Street, Rochester 4, New York.

Entry blanks for contributors to the Exhibition may be secured from Mr. H. Lou Gibson, 343 State Street, Rochester 4, New York. The entry fee is $1.00 in U. S. funds.

The last day for receiving entries is August 1, 1947.

CLINICAL CONFERENCE OF MID-WESTERN RADIOLOGISTS

The Seventh Annual Clinical Conference of Mid-Western Radiologists was held at the Statler Hotel, Cleveland, Ohio, on February 14 and 15, 1947. This meeting was sponsored by the Cleveland Radiological Society. Miscellaneous subjects were presented mainly by clinicians in the Cleveland area. More than one hundred members were in attendance and from every indication the meeting was a great success.

A fellowship hour and an informal banquet were held on Friday evening. Dr. Otto Glasser presented an interesting talk with lantern slide demonstration of cartoons depicting the layman's conception of the use of roentgen rays from the earliest times until the present.

Dr. Irving Cowan, President of the Milwaukee Roentgen Ray Society, extended an invitation to the Conference to meet in Milwaukee, Wisconsin, next year. This was accepted by the membership of the Cleveland Radiological Society.

The program of the meeting was as follows:

Friday Morning

Address of Welcome. Edgar P. McNamee, M.D., President, Ohio State Medical Association; Harry Hauser, M.D., Chairman; Donald D. Bannon, M.D., Assistant Chairman.

Diagnosis of Activity in Pulmonary Tuberculosis. R. C. McKay, M.D., Associate Professor of Medicine, Western Reserve University.

Intrathoracic Manifestations of Hodgkin's Disease. S. E. Wolpaw, M.D., Assistant Clinical Professor of Medicine, Western Reserve University.

Early Bronchial Obstruction. S. O. Freedlander, M.D., Associate Clinical Professor of Surgery, Western Reserve University.

Surgical Lesions of the Esophagus. E. B. Kay, M.D., Senior Clinical Instructor of Surgery, Western Reserve University.

Pneumoperitoneum and Phrenic Paralysis in Treatment of Tuberculosis. R. H. Browning, M.D., Assistant Clinical Professor of Medicine, Western Reserve University.

Differentiation between Atrial Septal Defect and Patent Ductus Arteriosus. A. D. Nichol, M.D., Department of Medicine, St. Luke's Hospital.

Differential Diagnosis of Coronary Artery Disease. R. W. Scott, M.D., Professor of Clinical Medicine, Western Reserve University.

Friday Afternoon

Fifty Years of Atomic Physics. Otto Glasser, Ph.D., Research Division, Cleveland Clinic Foundation.

Instruments for Measuring Radiant Energy. J. A. Victoreen, President, Victoreen Instrument Company.

Atomic Energy in Medicine. H. L. Friedell, M.D., Associate Professor of Radiology, Western Reserve University.

The Application of Radioactive Isotopes in Medicine. V. Lorber, M.D., Associate Professor of Biochemistry, Western Reserve University.

Round Table Discussion. Otto Glasser, Ph. D., Moderator.

Factors in Blood Formation. R. L. Haden, M.D., Division of Internal Medicine, Cleveland Clinic.

Chronic Monocytic Leukemia. R. W. Heinle, M.D., Assistant Professor of Medicine, Western Reserve University.

Clinical Diagnosis of Lymphoblastomas. S. M. Goldhamer, M.D., Teaching Fellow in Medicine, Western Reserve University.

Classification of Lymphatic Tumors. R. Dominguez, M.D., Director of Laboratories, St. Luke's Hospital.

Saturday Morning

Tantalum Cranioplasty. W. J. Gardner, M.D., Department of Neurosurgery, Cleveland Clinic.

Radiation Therapy in Arthritis. W. S. Duncan, M.D., Department of Orthopedic Surgery, St. Luke's Hospital.

Pneumoarthrography of the Knee. W. H. McGaw, M.D., Senior Clinical Instructor in Orthopedic Surgery, Western Reserve University.

Treatment of Fractures of the Transverse Processes of the Lumbar Spine. P. H. Kennedy, M.D., Medical Supervisor of the Youngstown Sheet and Tube Company.

Early Diagnosis and Management of Carcinoma of the Colon. T. E. Jones, M.D., Division of Surgery, Cleveland Clinic.

Treatment of Radiation Injuries of the Skin.

D. M. Glover, M.D., Associate Clinical Professor of Surgery, Western Reserve University.

Management of Functional Bleeding at the Menopause. G. B. Hurd, M.D., Department of Gynecology, St. Luke's Hospital.

Pelvimetry and the Obstetrician. H. P. Taylor, M.D., Department of Obstetrics, St. Luke's Hospital.

Heberden's Nodes: Clinical and Roentgenological Features of Degenerative Joint Disease of the Fingers. R. M. Stecher, M.D., Assistant Professor of Medicine, Western Reserve University.

Saturday Afternoon

Roentgenographic Diagnosis of Adrenal Tumors. J. J. Joelson, M.D., Associate Clinical Professor of Genitourinary Surgery, Western Reserve University.

Emphysematosis of the Urinary Tract. J. S. Lewis, M.D., Urologist, Youngstown Hospital Association.

Pulmonary Lesions in Q-Fever. C. H. Rammelkamp, M.D., Assistant Professor of Medicine, Western Reserve University.

Results of Vagotomy in Peptic Ulcer. W. C. McCally, M.D., Assistant Professor of Medicine, Western Reserve University.

Carcinoma Cervix Uteri: Five Year Results in 790 Cases. L. A. Pomeroy, M.D., Senior Clinical Instructor in Gynecology and Radium Therapy, Western Reserve University.

Preoperative and Postoperative Care in Gastric Surgery. F. M. Barry, M.D., Senior Instructor in Surgery, Western Reserve University.

Venous Thrombosis and Pulmonary Embolism. L. N. Atlas, M.D., Senior Clinical Instructor in Surgery, Western Reserve University.

Hyperparathyroidism and Hypoparathyroidism. E. E. Beard, M.D., Assistant Clinical Professor of Medicine, Western Reserve University.

Hyperparathyroidism and Hypoparathyroidism. E. P. McCullagh, M.D., Section on Endocrinology, Cleveland Clinic.

LOUISVILLE RADIOLOGICAL SOCIETY

The Louisville Radiological Society held its first post-war meeting on March 20, 1947, with an attendance of seventeen. Dr. Sydney E. Johnson was elected Chairman and Dr. Everett L. Pirkey, Secretary-Treasurer. It was decided to hold meetings on the second Friday of each month at the Louisville General Hospital. Cases were presented by the Staff of the Department of Roentgenology of the Louisville General Hospital.

The second monthly meeting of the Society was held at the Louisville General Hospital on Friday evening, April 11, 1947, with twenty members present. The formal portion of the program was presented by Drs. Joseph C. Bell and James Douglas on "The Roentgen Diagnosis of Lesions of the Cervical Spine" which was very illuminating, and was partly based on Dr. Bell's experiences while Chief of the Department of Roentgenology at Percy Jones General Hospital while in the Army of the United States. This was followed by the showing of interesting and unusual cases by the various members.

The next meeting will be held on May 9, 1947, at the Louisville General Hospital.

PERSONAL ITEMS

The many friends of Prof. Dr. Med. G. Fedor Haenisch, who is an honorary member of the American Roentgen Ray Society and was the Caldwell Lecturer at the meeting of the Society in Atlantic City in 1931, Dr. Leopold Jaches being President, will be glad to learn that he has survived the war, though having lost all of his personal possessions. Dr. Haenisch is now living at (24) Hamburg-Wandsbeck 1, Stephanstrasse 156, Germany, and he is particularly anxious to hear from his old friends in the American Roentgen Ray Society.

A recent letter from Prof. Dr. Med. H. Holthusen, Hamburg 20, Goernestrasse 29, Germany, who is also well known to all roentgenologists, states that he is continuing his work at the St. Georg Hospital, and contains the following paragraphs of general interest:

Recently I had the news that Prof. Alban Köhler died on February 26, 1947 seventy-two years old. I had a letter from him a fortnight ago. He seemed to feel well and had many plans.

Another of the pioneers has passed away. Their number has become very small in our country.

Not long ago we celebrated the eightieth birthday of Professor Walter. His cooperation with Albers-Schönberg in the field of roentgen technique will be remembered. He was a physicist and invented the water-cooled tube. He is active and is to be found at his bureau at the Physical Institute nearly every day.

NOTICE TO APPLICANTS AND DIPLOMATES OF THE AMERICAN BOARD OF RADIOLOGY

There are many Diplomates and candidates with applications on file whom we are unable to reach from their last known address. During and since the war many Radiologists have changed locations and now have a different address than appears in the records of the Board kept in the Secretary's office.

It is very important that these records be accurate and it is therefore urged that all those Diplomates whose present address is different from that which appears in the new Third Edition of "The Directory of Medical Specialists," and all applicants whose address has changed since they filed their application, notify the Secretary immediately.

B. R. KIRKLIN, M.D., *Secretary*
American Board of Radiology
Mayo Clinic, Rochester, Minnesota

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

HARVEY CUSHING: A BIOGRAPHY. By John F. Fulton. Cloth. Price, $5.00. Pp. 754, with 150 illustrations. Springfield, Illinois: Charles C Thomas, 1946.

Harvey Cushing (1869–1939) truly was a great man. He will be known as a teacher, author, investigator, medical historiophile, prodigious worker, acquaintance of many famous contemporary medical scientists, and stimulator of all with whom he came in contact. Undoubtedly he was the foremost graduate of the Harvard Medical School, and was certainly one of the most distinguished members of the faculty of the entire University. Unique is the fact that he was the author of one of the finest medical biographies of all time, and the subject of another which, in my opinion, rates close to it in interest, presentation and importance. John Fulton, Sterling Professor of Physiology in Yale Medical School, author of the biography, although much younger than Cushing, was long his intimate friend and an ideal biographer for him. He has the professional knowledge and the flair for writing in an effective, clear and engaging manner. Greatest of all is his energy, enthusiasm and ability to sift and evaluate a tremendous amount of material relating to his subject, thus shaping it into an authoritative and readable volume.

One hundred years from now and longer, Harvey Cushing will be remembered. This means that he has left a permanent mark, not only in this country but throughout the world where medicine is practiced. His name will live first as *the* creator of neurosurgery. It simply did not exist before his time. By rendering relatively safe the exploration of the cranial cavity, and showing that it was possible by special methods to effect surgical changes in other parts of the nervous system, such as the spinal cord and the gasserian ganglion, he brought to the attention of the profession and the world, the possibilities of this highly specialized branch of surgery. There is no better index of his lasting contributions to the welfare of mankind than the fact that he reduced the mortality in brain tumor operations from the appalling level of over 50 per cent to well below 10 per cent. For accomplishing this, he has a just claim to eternal greatness.

Apparently it was not that erratic genius William Stewart Halsted, the first Professor of Surgery at Johns Hopkins University, who directed him into this channel, for it was he who suggested that he enter the field of orthopedic surgery. Undoubtedly the inspiration preceded his association in Baltimore by some years. Evidence of his interest in the nervous system is shown by his enthusiastic dissection of the frog's nervous system when an undergraduate at Yale, the small statue in his rooms when a medical student, with the peripheral nerves marked on the surface in ink, and taking the first roentgenogram of a foreign body in the nervous system in the case of "Lizzie W." who entered the emergency ward on Cushing's service in 1896, having been shot in the neck by her bartender husband during a family brawl. These and perhaps other incidents may have played a role in his deciding to enter this then discouraging field. He disclosed the *real determining factor*, however, in later life at a dinner. given in Boston in 1926 for E. Wyllys Taylor. At this time he recalled the case of meningiomatous John Maloney, whom he had seen with Taylor in 1895 when he was a house pupil at the Massachusetts General Hospital. Cushing said on this occasion, "It made a great impression on me and started my interest in neurosurgery."

All will agree that Cushing has left as a heritage an outstanding group of men who are thoroughly trained in this intricate and highly specialized division of surgery. They are competent to carry his brilliant torch throughout this generation, and through their pupils in an endless procession thereafter.

He will not be remembered as a great teacher of undergraduate medical students. He was the type which is appreciated much more by the student in retrospect, some years after graduation from medical school. Dr. Cushing was not disposed to supply the student with a large amount of predigested factual information which he was expected to memorize. This is too

often the regrettable technique of some present day medical teachers. On the contrary, he had sound ideas of medical education which is not always immediately pleasing or acceptable to the student, namely, " . . . is not the essential thing to inculcate in the student's mind the habit of acquiring clinical knowledge for himself?"

Cushing's biography of William Osler which was undertaken in the latter part of his own life was a monumental accomplishment. Great it was because he was writing about the medical man of the century, and because he produced a classical biography which will always be regarded as one approximating perfection. His technique, seemingly of permitting Osler to tell his own story, is unsurpassed. As a biographer, however, he has submerged himself almost too much for such an intimate friend. This is not the easiest way of preparing such a contribution, although superficially it may appear to be. This method required a highly developed critical sense, good judgment and an intuitive dramatic instinct to weld such a fascinating story out of a myriad of letters, short notes scribbled on bits of paper, personal reminiscences, formal addresses, and the apocrypha which hazily surrounds almost all prominent men.

It is interesting to note that Fulton has employed the same technique of biographical writing which he has mastered in a manner entirely comparable to Cushing. Both Osler and Cushing were ideal subjects for a biographical study as they left multitudinous records of all kinds. It has been inferred by some that Cushing anticipated from an early date that he would be the subject of a biography and hence at a young age began preserving items which he thought would be pertinent. This, I doubt. It should be noted, however, that he did set aside in his will a sum of $5,000.00 if it were felt "that the publication of my diary might be of interest or help to medical students."

The research activities of Cushing and his associates are of primary importance but, like the work of many other great men, his claims have not always been supported by subsequent investigators. Nevertheless, he did make substantial contributions and stimulated thought along the lines of nervous system pathology and physiology which will be of lasting importance. The Cameron Lectures, given in England in 1925, epitomize his contributions. The three addresses were as follows: "The Third Circulation and its Channels," "The Pituitary Gland as now Known," and "Intracranial Tumors and the Surgeons."

Harvey Cushing will always be known as a versatile, brilliant, accomplished surgeon, an erudite scholar and bibliophile, a teacher and developer of younger man, a tireless worker, who was sometimes impetuous and on rare occasions amazingly naïve. His sterling qualities so far exceeded those of a less desirable nature that the latter may now be forgotten. Sometimes, in fact, those that seemed so serious at the time may be nullified by at least a faint smile. Of these, and this is said with all reverence, his controversy with the protectors of the Boston Common during the first world war, now appears rather trivial. I say this with all respect, however, because one attribute which has made Boston great is the willingness of its citizens to give all for a principle.

This volume in the opinion of the reviewer is a classic. Along with "The Life of William Osler" it might well serve as a model for future medical biographies. It is a painstaking, exact, detailed, carefully written and verified record of one of the notable geniuses of the medical profession. Every medical student could read and reread it with profit, as could all members of the profession. Furthermore, it would be of value to all persons who have a desire to obtain more information, from a new angle, about the development of our country between the years of 1869 and 1939, a period of our greatest growth and development. It is excellent contemporary history, as it tells of Yale University in the late eighties; of Harvard Medical School shortly thereafter, and the first class in that institution which was given four years' instruction instead of three; of the remarkable development of the medical department of Johns Hopkings University and the illustrious group of the then young men who gave that institution such an impetus; of the initial organization of the Peter Bent Brigham Hospital and the relation to its parent institution the Harvard Medical School from 1912 to 1935; of his many journeys to England and the continent, and his widespread acquaintance with notable men, chiefly medical and surgical; and, finally, the story of his interesting days during his retirement at Yale University.

Cyrus C. Sturgis

The X-Ray Treatment of Accessible Cancer. By D. Waldron Smithers, M.D., D.M.R., Director of the Radiotherapy De-

partments The Royal Cancer Hospital (Free) and The Royal Free Hospital; Radiotherapist Brompton Hospital for Diseases of the Chest; Associate Physician for Radiotherapy West End Hospital for Nervous Diseases, etc. Cloth. Price, $8.50. Pp. 147, with 102 illustrations. Baltimore: The Williams & Wilkins Company, 1946.

For some time the need for an adequate presentation of this subject has been increasingly felt. Smithers has undertaken to supply this need and has done it well. The book is well printed on good paper, and many excellent photographic cuts, some in color, add greatly to the value of the text. The book is divided into eleven chapters dealing with: (1) general principles of treatment of accessible cancer; (2) apparatus; (3) classification of accessible cancer, staging, records and assessment of results; (4) dosage and biological response of tumors to irradiation; (5) cancer of the skin; (6) cancer of the lip, nose, ear, eyelids and cornea; (7) cancer of the external genitalia; (8) cancer of the mouth; (9) cancer of the cervix uteri; (10) roentgen treatment of cancer made accessible by surgical exposure; (11) treatment of lymph node metastases. In an appendix may be found definitions and isodose curves.

In the preface Smithers justly emphasizes the importance of early diagnosis, but he also stresses the need of a complete diagnosis. Too often, unfortunately, as soon as a label of "carcinoma" or "fibrosarcoma" has been attached to a malignant process, the problem of diagnosis is considered solved. A complete diagnosis should include knowledge, not only of the primary growth, its history and symptoms, but it should also include knowledge of secondary foci and mode of spread, because this knowledge when it can be obtained, may account for symptoms which are not accounted for by the primary tumor. A complete diagnosis should account for all the major symptoms.

Therapeutic radiologists will find, in chapter 4, much food for thought and perhaps for revision of methods of treatment. Smithers' discussion of the value of single and repeated biopsy, of serial biopsy, of the mode of action of radiation on malignant cells, and of the value of repeated counts of malignant cells in relation to treatment, is worthy of careful thought. Some of these procedures often cannot be employed routinely in institutions where private patients are cared for or in private practice; but it would appear that, when they can be used, they may furnish information which may greatly help in arranging the treatment and in understanding its effect. One of the outstanding features of the book is the brief, but adequate, presentation of short distance, low voltage treatment and of so-called contact therapy.

Both the author and the publishers are to be commended for bringing out this much needed book.

A. U. Desjardins

A Handbook of Radiography. By John A. Ross, M.A. (Camb.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.M.R.E. (L'pool). Visiting Radiologist, Alder Hey Children's Hospital, Liverpool; Hon. Radiologist, Warrington Infirmary, Warrington; Radiologist, General Hospital, Warrington, etc. Second edition. Cloth. Pp. 165, with 92 illustrations. London: H. K. Lewis & Co., Ltd., 1946.

As the title implies, this book was written for technicians and physicians or students interested in roentgen techniques. It reads easily and is relatively comprehensive.

Divided into fifteen chapters, it considers the roentgenography of each part of the body separately. In addition, the author has dedicated one chapter to the "care of the patient" which everyone interested in the field could read with benefit. A brief résumé of the physics concerned completes the work.

The illustrations are all line drawings. In many respects, they are inadequate and in some instances seemed misleading to the reviewer. Because the illustrations are not as good as those in several other roentgenographic texts, its sale will probably be limited unless the price more than compensates for this deficiency.

Philip J. Hodes

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

SKELETAL SYSTEM

Fischer, Frederick J., and VanDemark, R. E. Sagittal cleft (butterfly) vertebra. *J. Bone & Joint Surg.*, Oct., 1945, *27*, 695–698.

One hundred years ago von Rokitansky described a congenital division of the twelfth thoracic vertebral body into lateral halves with an intervening cleft. Since then, the literature dealing with this condition has not been abundant, particularly in English. The authors report 2 cases of this condition. In the first patient the third lumbar vertebra was affected; in the second, the eighth dorsal.

In the roentgenogram, the typical "butterfly" appearance is seen in the anteroposterior film where the wings of the butterfly are represented by the two symmetrical halves of the vertebral body. The two halves may be separated by a wide cleft, bridged by strands of bone, a bony bridge, or demarcated only by a narrow sagittal cleft between the two halves. In this typical picture, the halves are of equal size; however, they may be slightly unequal, displaced laterally or wedged anteriorly. Resultant scoliosis or kyphosis may be noted. The adjacent intervertebral spaces may be narrowed, but not collapsed. Compensatory changes may occur in the adjacent vertebrae, as if an attempt had been made to fill in the deficiencies in the form of an anomalous vertebra. Other vertebral and rib anomalies may be associated.

From an orthopedic standpoint, the authors call attention to two groups or types of this condition, the symptomatic and the asymptomatic. The symptomatic group are usually characterized clinically by the presence of mild, moderate, or severe local deformity occasionally followed by pain, and roentgenographically by anterior compression of the vertebral body halves, and less frequently, by inequality of the two vertebral halves or their lateral displacement. In these cases, the sagittal cleft roentgenographically distinguishes the anomaly from unreduced vertebral body fractures with anterior or lateral compression and lateral displacement. Clinically the symptomatology may resemble that seen in old unreduced fractures of the vertebral body. Certain asymptomatic cases are characterized clinically by the absence of deformity. Roentgenographically they show division, not always complete, of the vertebral body into two equal halves, without anterior compression or lateral displacement.—*R. S. Bromer.*

McMaster, Paul E. Unilateral hypoplasia of lumbosacral articular processes. *J. Bone & Joint Surg.*, Oct., 1945, *27*, 683–686.

McMaster reports a rare, anomalous, unilateral hypoplasia resulting in rudimentary lumbosacral articular processes and associated neural arch of the fifth lumbar vertebra. He states that no such case seems to be described in the literature.

The roentgenograms of his patient showed rudimentary opposing articular processes on the left side, between the fifth lumbar and first sacral segments. The left lamina of the neural arch of the fifth lumbar vertebra was also hypoplastic. The corresponding right articular processes and arch seemed to be normal. The spinous process of the fifth lumbar vertebra appeared normal, although some incomplete fusion of the neural arch of the first sacral segment was present. Narrowing of the fifth lumbar disc was not apparent. Moderate haziness of each sacroiliac articulation indicated arthritic changes.

A striking feature of the case presented was the fact the patient suffered no pain on the side of the anomaly but that he did have right low back pain which was apparently due to the abnormal stress and strain placed upon the right low back muscles, fasciae, and ligaments.

In the case presented, the anterior portion of the left half of the fifth lumbar arch, namely the pedicle and the superior articular process, appeared normal; while the posterior portion, including the lamina and the inferior articular process, was underdeveloped. Thus, it is likely that there were two centers of ossification for the left half of the neural arch, the one for the posterior being hypoplastic. Ossification of each

half of a neural arch, including the articular processes, is usually from one center. An explanation of the rudimentary left articular process of the sacrum appears to lie either in the hypoplasia of the center of ossification for the left half of the sacral arch, or in underdevelopment due to lack of stimulus from an apposing normally developing inferior process of the fifth vertebra. The latter appears likely, as the two apposing processes were very small and rudimentary and corresponded in size, shape, and approximation.—*R. S. Bromer.*

GENERAL

Rosenthal, Nathan, and Erf, Lowell A. Clinical observations on osteopetrosis and myelofibrosis. *Arch. Int. Med.*, June, 1943, *71*, 793–813.

The clinical and laboratory observations in 1 case of osteopetrosis and 17 cases of myelofibrosis are discussed by the authors.

Osteopetrosis is frequently found during childhood and the severe types are characterized by mental and physical retardation; weakness; atrophy of the optic nerve; hydrocephalus; poor dentition; and progressive enlargement of the spleen, liver and lymph nodes; progressive development of a refractory type of anemia (associated usually with thrombopenia or leukopenia and often with slight myelemia) and occasionally by osteomyelitis and pathologic fractures. It has familial and hereditary features and a history of consanguinity is frequent. The cause remains unknown.

Myelofibrosis is clinically characterized by weakness, dyspnea, refractory anemia, splenomegaly, periostitis and bone pains which are frequently deep seated (in the back and lower extremities). It occurs equally often in the two sexes and may be evident at birth or in late adult life. There are no familial or hereditary characteristics known. The prognosis for survival varies from five to six weeks when the disease is acute, to several years when it is chronic. The cause is unknown.

The cellular changes of the peripheral blood of patients with myelofibrosis and those with osteopetrosis are frequently similar. The peripheral blood picture occasionally resembles that of myeloid leukemia but more often that of a refractory type of anemia associated with leukopenia, thrombopenia and the presence of a few immature erythroid and myeloid cells.

The postmortem observations are usually hepatosplenomegaly and occasionally generalized lymphadenopathy, a sclerotic or fibrotic marrow and sclerosis or rarefaction of the osseous system. Microscopically the spleen, liver and lymph nodes often show chronic inflammatory changes with fibrosis, myeloid hyperplasia and extramedullary erythropoiesis. The marrow cavity may be filled diffusely with fibrous tissue or there may be areas of hemapoietic tissue interspersed in the fibrous tissue. The endosteal bony trabeculations are usually increased in size and number. A roentgen study of the bone is important for confirmatory diagnosis in all cases of osteopetrosis.

About 50 per cent of patients with myelofibrosis show, on roentgen examination, mottled rarefaction or irregular condensation of the cortical portions of the bone and splintering or elevation of the periosteum. A review of the literature shows that some authors describe roentgenologic changes of the bones in their cases while others present cases in which roentgen changes of the osseous system were not evident. Although in some cases the fibrosis extends from the endosteum to the periosteum, fractures rarely occur.

These conditions may be differentiated from refractory types of anemia, splenomegaly and leukemia by sternal puncture and conclusively by sternal biopsy.—*J. J. McCort.*

Farber, Jason E., and Margulis, Aaron E. Blue scleras, brittle bones and deafness. *Arch. Int. Med.*, May, 1943, *71*, 658–665.

This article concerns a family which has been affected with blue scleras, brittle bones and deafness through at least four generations in which there has been no consanguinity. The family is of Scotch-English lineage. A genealogic tree has been outlined accounting for 52 members. Twelve members of the family possessed one or all of the features of the disease. One has only blue scleras; 7 have both blue scleras and brittle bones; 4 have all three defects. Eight of the affected ones are males and 4 are females. Of 14 males exposed to the risk of inheriting the syndrome, 7 did inherit it. Of 10 females similarly exposed, 4 showed the syndrome. Thus, of 24 children of seven matings heterozygous in respect to the phenotype under discussion, 11 inherited the anomaly. Transmission by a male parent occurred in 2 instances and by a female in 2 instances. Transmission took place directly between succeeding generations. The inherited factor thus appears to be a mendelian dominant and not sex linked.

Three members of the group have been stud-

ied in detail. Roentgenograms of the skeleton revealed slender bones, generalized osteoporosis, and deformities, resulting from multiple fractures.

The authors believe that since it seems well established that there is osteoporosis in this disease the fact that there is lacking unequivocal chemical evidence of a negative chemical balance appears to be of lesser significance. A degree of calcium loss that could be determined with ease in necessarily short term metabolic studies would be incompatible with the longevity of life demonstrated in the afflicted members of this family. It is also believed that negative phases of calcium balance might occur only during certain restricted times in their lives and it would be a matter of chance that these should be discovered. The tendency to fracture seems to be a recurring cyclic affair and diminishes with increase in age. This might conceivably be associated with endogenous changes or it might be due to the varying exposure to risk of fracture at different times of life and the growing apprehension and personal solicitude that would come with maturity.—*J. J. McCort.*

ROENTGEN AND RADIUM THERAPY

Fowler, Edmund P. Irradiation of the eustachian tube—An anatomic, physical and clinical study of a treatment for recurrent otitis media applied to aero-otitis. *Arch. Otolaryng.*, Jan., 1946, *43*, 1–11.

Exposure of the eustachian tube to radon, radium or roentgen ray will often prevent recurrent otitis media when this condition is due to excess lymphoid tissue about the pharyngeal ostium of the eustachian tube. It is particularly valuable for certain cases of otitis media due to barotrauma (aero-otitis).

The known susceptibility of lymphoid tissue to irradiation is the basis for the use of radiation in the nasopharynx. This susceptibility was first observed in lymph nodes, but experience has shown that adenoid tissue is quite as sensitive. It is remarkable how little radiation is required to produce a profound effect. After one treatment, the germinal centers throughout the specimen have disappeared and the number of lymphocytes is greatly reduced. Many of those which remain have pyknotic nuclei. The capillaries in the irradiated tissue are prominent. Their endothelium is thickened and their walls are hyalinized. Connective tissue cells are much more prominent than in the control biopsy specimen.

In the European Theater of Operations 95 pairs of temporal bones have been removed from dead airmen and examined grossly preparatory to microscopic study. Eight pairs of bones have been removed from ground personnel of the same age group. This material shows that nasopharyngeal lymphoid tissue is common in American airmen in England. The tissue covers the torus tubarius in many airmen who never had enough aero-otitis to report to their group surgeon. Lymphoid tissue tends to make the pharyngeal orifice of the tube a narrow slit to which mucus adheres. To date, one bone from each of 16 persons has been cut and stained so as to allow analysis of the microscopic appearance of the entire length of the tube. Most interesting is the low grade eustachian salpingitis which is present in 11 of the 16.

The complete or partial clinical records of 80 airmen who had been treated with radon or radium were available for this study. Airmen are subjected to violent changes in pressure, and if they do not have normally functioning tubes, this is quickly apparent during descent or ascent. That 46 of the 80 airmen with recurrent aero-otitis from various causes are known to have returned to full flying duty after radiation treatment is significant. This means that in 57 per cent of all the cases there was marked improvement.

The patients were treated with two types of platinum applicators, one type containing radon and the other radium element. The earlier patients were treated with a platinum applicator having a wall 0.65 mm. thick and containing radon. The later patients were treated with capsules having a wall 0.5 mm. thick and containing radium sulfate. The earlier patients were treated with 33 milligram-hours to each side of the nasopharynx, and it was found that four to six treatments at three week intervals were necessary. The later patients received 50 milligrams to each side of the nasopharynx for two to four treatments at four week intervals.

Results comparable to those obtained with radon and radium have been obtained with roentgen rays. In using this type of therapy it has been thought unwise to give more than 100 r to each side at a sitting.—*Mary Frances Vastine.*

Cutler, Max. Cancer of the larynx; five year results of concentration radiotherapy. *Arch. Otolaryng.*, April, 1946, *43*, 315–330.

This is a report on 119 consecutive patients

with cancer of the larynx who were treated by irradiation in the Chicago Tumor Institute from the time it opened in April, 1938, through December, 1942.

Concentration Radiotherapy. The basic principle is that large daily doses are given over a comparatively short period. The total dose is sufficient to cause an "epithelite" and usually also an "epidermite." With certain exceptions, the dose is increased daily and the size of the cutaneous portal is diminished. Concentration radiotherapy is much more intense than the usual divided dose technique known as the Coutard method. Instead of 6,000 to 7,000 r being given in four to seven weeks to two fields (the Coutard method), 6,000 to 6,500 r is administered in eighteen days to one field.

Voltage—Roentgen Intensity and Fields. The higher voltage appears to have advantages. Most of the patients are treated with 400 kilovolt roentgen rays. The roentgen intensity has varied between 5 and 10 r per minute. Except on special indications, one lateral field is used. Because the central remnant of the regressing carcinoma is its most radioresistant portion, the field is reduced gradually as the daily dose is increased.

Time (Total Number of Days of Treatment). Within certain still undefined limits, the more radioresistant the lesion, the shorter must be the time of treatment and the larger the daily dose. A superficial papillary carcinoma of the true cord, for example, can be sterilized with 6,000 r being given through two portals and divided equally over a period of thirty to forty days. A carcinoma of the true cord that has infiltrated the muscle and caused complete fixation, as a rule, cannot be sterilized by this method but requires more intensive irradiation in the form of larger daily doses, 6,000 r being given through one portal in eighteen days or less.

Radium Treatment—Telecurietherapy. The apparatus for telecurietherapy of the Chicago Tumor Institute contained 10 gm. of radium until September, 1943, when 3 gm. was released for war purposes. The skin distance is 12.5 cm., the filter 1 mm. of platinum and the portal 6 by 6 cm. No definite conclusions can be stated as to the comparative value of roentgen rays and radium.

Present Technique. The following two roentgen-ray techniques are in general use in the Chicago Tumor Institute:

1. For an early intrinsic carcinoma of one or both true or false cords with complete or partial mobility: 400 kv.; 5 ma.; filter, 5 mm. of copper; distance, 84 to 115 cm.; single lateral port, 5 by 5 cm. gradually diminished to 3 by 3 cm.; average intensity, about 7 r per minute; rate of treatment, two equal treatments daily for eighteen days, beginning with 100 r twice a day and increasing to 250 r twice a day; total dose, 6,000 r measured on the skin.

2. For more extensive intrinsic and for extrinsic carcinoma with or without regional cervical metastases and with partial or complete fixation: 400 kv.; 5 ma.; filter, 7 to 8 mm. of copper; distance, 75 to 80 cm.; single lateral port, 7 by 5 cm. gradually diminished to 4 by 3 cm.; average intensity, about 6 r per minute; rate of treatment, two equal treatments for eighteen days, beginning with 85 r twice a day and increasing to 275 r twice a day; total dose, 6,500 r measured on the skin. When the metastases are too extensive to be included in the fields, additional irradiation is given to such areas.

The most effective telecurietherapy technique has been the following: total dose, 72,000 milligram-hours to each of two lateral fields; radium content, 7 to 10 gm.; distance, 12.5 cm.; field, 6 by 6 cm.; rate of administration, two treatments daily for twelve days, the dose being increased from 2,000 mg-hr. to each side daily to 10,000 mg-hr. to each side daily.

Conclusions. Modern radiation therapy is an increasingly effective method of treatment for laryngeal cancer. Forty-two per cent five year cures and 39 per cent three year cures were obtained in a series of 118 unselected cases. Concentration radiotherapy has resulted in the curing of certain forms of cancer of the larynx hitherto regarded as incurable by irradiation. When the cords are freely movable or only partly fixed by the carcinoma, curability is high, but when they are completely fixed, cure is less common and laryngectomy is indicated. The most important result of this research is the demonstration that a group of patients with intrinsic squamous carcinoma of the larynx too advanced for laryngofissure and requiring total laryngectomy have been cured by irradiation. As the results of irradiation improve, the field for operation becomes smaller. Laryngectomy should be limited to intrinsic lesions with complete fixation of the cords occurring in good surgical subjects of good life expectancy. It is reasonable to expect that further improvements in the technique of irradiation and earlier diag-

nosis will further diminish the necessity of total laryngectomy.—*Mary Frances Vastine.*

Proctor, Donald F. Irradiation for the elimination of nasopharyngeal lymphoid tissue. *Arch. Otolaryng.*, May, 1946, *43*, 473–480.

In many patients irradiation of nasopharyngeal lymphoid tissue is essential for its adequate elimination. Lymphoid tissue is so sensitive to irradiation that minute dosages will cause its regression and eventual disappearance without damage to surrounding tissues.

The active part of the applicator used in the irradiation of the eustachian tubes is a Monel metal tubular chamber 15 mm. in length, with an inside diameter of 1.5 mm., a wall thickness of 0.3 mm. and an outside diameter of 2.1 mm. The chamber contains 50 mg. of radium sulfate.

Practically all the beta rays emitted by this applicator are absorbed by the tissues of the nasopharynx. About 75 per cent of the beta rays are absorbed in the first 3 mm. of tissue, but 6 to 7 per cent of the hard beta rays are still detectable after passing through 20 mm. of tissue. In contrast, less than 10 per cent of the gamma rays are absorbed. The effects aimed at and obtained by radiation therapy are entirely dependent on ray absorption, and are roughly proportional to the amount of absorption.

Twenty-five days is the interval between treatments. As a rule, three treatments are adequate.

The dosage that has been found most effective with this applicator is "1 gm. twenty seconds" on each side of the nasopharynx—since this applicator contains only 50 mg. or one-twentieth of a gram, the dosage is twenty times as long—400 seconds or 6 minutes and 40 seconds, on each side.

Technique of Treatment with the Radium Applicator.

1. The applicator should be left in the lead cylinder until one is ready to insert it into the patient's nasopharynx. A lining tube of glass may be fitted into the hole in the lead cylinder and kept filled with 95 per cent alcohol, which is a convenient means of sterilizing the applicator. Heat must never be used.

2. The patient should lie comfortably on a couch during the treatment.

3. The alarm time clock should be used to control the duration of the treatment.

4. Remove the applicator from the lead cylinder, wash off the alcohol with water, dip into a lubricant (one that is water soluble so that it may be quickly washed off at the conclusion of the treatment) and insert into the nasopharynx.

5. In placing the applicator, the middle of the radium-containing chamber should be in direct contact with the tissue in the fossa of Rosenmüller or the orifice of the eustachian tube. The exact location and the depth from the external nares to the area to be treated can be determined only by a preliminary examination with the nasopharyngoscope in one side of the nose and a calibrated rod or applicator in the opposite side.

6. The most important protection for personnel using the treatment is distance. The intensity of the radiation varies inversely with the square of the distance. During the treatment, all personnel who are constantly handling the applicator should be at a distance of 25 or 30 feet.

7. At the conclusion of the treatment, the mucus adhered to the applicator should be removed with a brush fixed to the table or the sink and the applicator quickly replaced in the lead cylinder.

8. The nasopharynx is re-examined at the end of three to four weeks and treatment repeated at this interval and subsequent intervals until satisfactory regression has occurred.—*Mary Frances Vastine.*

Boies, Lawrence R. Irradiation of nasopharyngeal lymphoid tissue; an evaluation. *Arch. Otolaryng.*, Aug., 1946, *44*, 129–140.

In 1939 Crowe and Baylor renewed the interest of physicians in nasopharyngeal lymphoid tissue. Their first report and subsequent ones of their co-workers indicated that:

1. There is in children eight to fourteen years of age a high incidence (nearly 40 per cent) of impairment of thresholds for some or all of the tones above C_4; i.e., for frequencies higher than 2,048 cycles per second.

2. A common cause of this early hearing loss is to be found in catarrhal changes of the middle ear which originate from infected lymphoid tissue in the nasopharynx.

3. Hyperplasia of nasopharyngeal lymphoid tissue is common in children. A majority of those who have had their tonsils and adenoids removed will, when examined, be found to have adenoid masses or hyperplasia of the lymphoid tissue in the fossa of Rosenmüller and in the

region of the nasopharyngeal orifice of one or both eustachian tubes.

4. Irradiation of this tissue with a 2 gram-minute dose of radon to each side is an effective method of causing regression of a small amount of the lymphoid tissue. This amount of irradiation has no harmful effect on the nasopharyngeal structures.

5. Children with impairment of hearing for high tones could have lesions other than those secondary to tubal occlusion caused by excessive lymphoid tissue. Treatment in all cases is advisable, however, as a therapeutic test if the nasopharynx is not normal in appearance.

6. The most immediate benefit from radon therapy was found in those children who had impaired hearing for all tones or difficulty in understanding the spoken voice.

7. Rechecking every few months is important.

In July, 1943, the author first used radon filtered so as to give the more caustic beta rays. Three hundred millicurie-minutes (0.33 gram-minute) was used, and at the same time 2,000 millicurie-minutes (2 gram-minutes) of gamma radiation was used on the opposite side. Clinically no difference in effect was noted. The total number of roentgens received by the lymphoid tissue is the same at a depth of 2.25 mm. whether one is employing 2 gram-minutes of gamma radiation or 0.33 gram-minute of beta and gamma radiation. At a greater depth, such as 5 mm., the roentgen dose is about 2.6 times greater with the gamma radiation than it is with the combined beta and gamma radiation, but the dose of either is very small at this depth.

Summary. Small doses of radium or radon filtered so as to yield chiefly the beta rays seem effective in controlling the pathologic effect of small amounts of this tissue in its relation to the normal function of the eustachian tube. The dose is so small that no local reaction is observed or experienced in the form of hyperemia, irritation or other untoward effect. Crowe regards this action as a "biological" one in which mitosis of the lymphocyte is inhibited.—*Mary Frances Vastine.*

Watson, B. P., and Gusberg, S. B. Prevention and treatment of carcinoma of the vulva. *Am. J. Obst. & Gynec.*, Aug., 1946, *52*, 179–190.

Symptoms and Prevention. Any irritated or ulcerated lesion which does not yield within a very short time to simple therapy should be subjected to biopsy by free excision.

Every leukoplakic area which causes constant itching, necessitating scratching for its relief—with consequent breaking or fissuring—should be excised.

The average age of the patients with vulvar cancer was fifty-nine. This is, in general, a disease of advanced years.

The average delay from the onset of vulvar symptoms to treatment of the cancer in the authors' group was three and three-fourths years. Pruritus was notably the symptom with the longest duration—an average of 8.1 years.

Treatment and Results. The operation of radical vulvectomy with bilateral dissection of the superficial inguinofemoral nodes is an efficient one.

The use of wide local excision as a biopsy method is proposed; its therapeutic value in low grade malignancies of the vulva is suggested.

The prognosis is grave if there is extension of the disease into the vagina.

The authors are not as pessimistic as many observers concerning the general group of vulvar carcinomas. Many of them can be cured.

The authors have discarded irradiation as anything but a palliative treatment in carcinoma of the vulva. They do have one patient in their series who had an extensive lesion and was treated by partial vulvectomy, radium needles locally, and roentgen therapy. This is the only patient with vaginal involvement who survived.—*Mary Frances Vastine.*

Gray, Laman A., Friedman, Milton, and Randall, William S. Observations on the treatment of adenocarcinoma of the uterus. *Surg., Gynec. & Obst.*, April, 1946, *82*, 386–404.

In the past three years, 10 women with adenocarcinoma of the uterus, comprising 0.5 per cent of the patients on the gynecologic service, have been treated at Walter Reed General Hospital. These cases are presented without five year follow-up for two reasons: (1) residual carcinoma was found in 6 of the 7 uteri which had received enormous doses of radium, and (2) the presence of ovarian metastasis in 3 patients emphasizes the necessity for oophorectomy as part of the treatment.

Analysis of the Cases. In this series of 10 cases of adenocarcinoma of the uterus, 9 were

moderately advanced or advanced but without induration of the pelvic ligaments. They were, therefore, considered inoperable.

Carcinoma protruded from the cervical os in two uteri and ovarian metastasis was subsequently found in 3 patients.

Microscopic examination revealed moderate differentiation (Grade 2) in 4 cases, moderate undifferentiation (Grade 3) in 4 cases, and complete undifferentiation in 2 cases. This distribution is atypical in that most large series have shown the majority of cases to be differentiated adenocarcinoma.

In 7 of the 10 cases, treatment with radium was followed by panhysterectomy and bilateral salpingo-oophorectomy. The radium dose ranged from 6,030 to 12,480 milligram-hours. The size of the uterus was moderately or greatly reduced following radium.

The operative procedure was not unduly difficult except in 1 case in which the residuals of pelvic inflammation complicated the operation. In every instance the parametrial tissues were somewhat sclerotic with lack of resiliency and the sutures had to be tied with care. The uterine vessels seemed smaller than usual.

Apparently viable cancer cells were found in the superficial myometrium in 6 of the 7 excised uteri. There was deeper extension in three. The 1 uterus in which no residual carcinoma was found had a highly malignant, undifferentiated adenocarcinoma which received 12,480 mg-hr. of radium given in two exposures.

The location of residual carcinoma deep in the myometrium explains why probatory curettage performed several months after radium treatment in order to evaluate its effectiveness may fail to extract the residual carcinoma.

It is possible that, after irradiation, some of this residual carcinoma might not have mainfested itself clinically for five years or longer. However, hysterectomy is necessary to complete eradication of the local tumor.

Reasons why the combined radium-surgical treatment gives the best results, according to the literature, include: (1) reduction of intrauterine infection preoperatively, (2) reduction in size of the uterus simplifying the operative procedure, (3) immobilization of cancer cells in the uterus reducing metastasis, (4) sclerosing of lymphatics and blood vessels, reducing metastasis, (5) prevention of recurrence from operative spill in the vagina and abominal incision, and (6) favorable results born e out by statistical evidence.

Statistical evidence, however, is the only concrete information on the value of preoperative irradiation. The available statistics are based on too small series to be conclusive. Series of as many as 100 cases with uniform radium treatment in doses of 4,000 mg-hr. or more, should be available before the role of preoperative irradiation can be evaluated.

Preoperative roentgen therapy has been recommended by some who emphasize the additional destruction of tumor cells in the parametrium by this method. However, the intensive roentgen therapy necessary to give a lethal tumor dose may cause destructive changes in the bowel and adhesions. The immediate postradium complications following the use of large doses were not serious in this particular series. However, the later deleterious effects of intrauterine radium in the large doses employed in this series are not to be overlooked. Postoperative hospitalization is lengthened. Recovery of vitality is often delayed for months. There may be delayed healing of the vaginal wound, intermittent diarrhea, and frequency of urination. If the eventual cure rate is increased, these disadvantages can be disregarded.

Three patients in this series had metastasis to the ovary. This not uncommon incidence of ovarian metastasis is frequently overlooked in planning therapeutic procedures for adenocarcinoma of the uterus. It constitutes an important argument against the use of radium alone in the treatment of this disease. Preoperative radium cannot materially affect the metastatic lesion in the ovary. Consequent postradium hysterectomy should not be unduly delayed.

Conclusions.

1. This study suggests that radium alone will not completely eradicate adenocarcinoma of the uterus. Subsequent excision of the uterus, tubes and ovaries is necessary.

2. The exact role and specific indications for preoperative radium therapy have not yet been satisfactorily delineated.—*Mary Frances Vastine.*

Stowe, Lyman M. A histologic study of the effect of irradiation on adenocarcinoma of the endometrium. *Am. J. Obst. & Gynec.*, Jan., 1946, *51*, 57–66.

Since it is likely that complete eradication of the tumor by whatever means of therapy is employed is essential to the production of cure,

it seems logical to examine the effect of radiation therapy on the carcinomatous tissue. The purpose of this paper is to present a series of 53 cases of adenocarcinoma of the body of the uterus, which were proved by examination of tissue obtained by curettage, and which have been treated either by a combination of radium application and surgery, or by radium, deep roentgen therapy, and surgery. The material for histopathologic study was obtained by subjecting the patients to panhysterectomy with bilateral salpingo-oophorectomy at approximately five weeks after the completion of radiation therapy. The gross material has been carefully re-studied and multiple blocks of tissue from each uterus were sectioned.

Summary and Conclusions.

1. The literature dealing with the detection of residual tumor and its frequency in adenocarcinoma of the endometrium treated with radium, radon, and deep roentgen therapy has been reviewed. These reports show persistence of apparently viable tumor in from 12.5 to 89 per cent. There was wide variation in the amount of therapy given, in the type of radium or radon applicators used, in the intervals between radiation therapy and operation, and in the thoroughness of the search for residual tumor in the operative specimens.

2. The incidence of residual tumor in the uteri of the 53 patients studied in this paper was 50.9 per cent. All of these patients had intrauterine radium application; 23 had deep roentgen therapy as well. In a small series where roentgen therapy was employed in adequate dosage together with radium irradiation, the incidence of residual tumor fell to 40 per cent, while in a longer series where radium alone was used the incidence was 56.5 per cent.

3. An applicator designed to overcome the effect of variations in size and shape of the uterine cavity is described. It was used in 46 of the 53 cases.

4. In the majority of instances where residual carcinoma was found, it was seen in the musculature of the uterus.

5. In view of the fact that tumor persisted after irradiation in a high percentage of cases, and especially in view of its frequency in the myometrium, it is concluded that surgical attack must be considered to be the essential feature of curative therapy in the treatment of adenocarcinoma of the corpus uteri. It is still possible that preliminary irradiation may be useful as additional therapy or as palliation.—*Mary Frances Vastine.*

BUSCHKE, FRANZ, and CANTRIL, SIMEON T. Indications for roentgen therapy of bladder carcinomas. *Surg., Gynec. & Obst.*, Jan., 1946, *82*, 29–35.

Of 68 patients with advanced carcinoma of the bladder treated between 1934 and 1940 with 800 kilovolt roentgen irradiation, 10 are so far clinically well and cystoscopically without evidence of disease. Seven of these are well and have remained well for more than four years. The analysis of the successes and failures have led to certain tentative conclusions with regard to indications and contraindications of this procedure.

Contraindications. (1) Poor general condition of the patient; (2) poor bladder drainage and capacity; (3) marked infection; (4) previous suprapubic operation particularly in the presence of any obstruction or infection; (5) cancers inoperable because of extravesical extension.

Indications. (1) Extensive papillary carcinomas in which infiltration cannot be demonstrated. (2) If papillary carcinomas recur after repeated fulguration or if the surgeon feels that a certain type of carcinoma will probably recur after fulguration, roentgen therapy is indicated without further delay (if it is considered at all). (3) While there is some indication that certain infiltrative types of bladder carcinoma might be cured by roentgen therapy, the likelihood of such a cure becomes practically nil when the bladder muscle is invaded. (4) If the infiltrating lesion is so located that a complete bladder resection is the only surgical procedure possible, interstitial irradiation, preferably in the form of implantation of removable radium needles, is probably superior to roentgen therapy (provided the actual size of the tumor is not beyond 3.5 cm. in diameter).

Technique of Treatment.

It is necessary to include the entire bladder in the field during the whole course of treatment. Furthermore, it seems necessary to treat the entire bladder in the recurrent papillary growth (the most suitable type for roentgen therapy) in order to prevent recurrences in other parts of the mucosa.

Necrotic ulcers in the bladder, which are at times found after treatment of uterine carcinoma, have never been observed in roentgen

therapy of bladder carcinoma. This indicates that they are most likely due to faulty technique in the radium application in cancers of the cervix rather than to the external irradiation.

Most of the patients are treated through three fields, one central anterior field, and two oblique lateral fields centered toward the bladder. (In this way the rectum receives its main irradiation through the anterior field only, while most of the rectum is spared by the posterior fields. Only occasionally has even a transitory diarrhea been observed.)

The use of supervoltage roentgen therapy in the treatment of bladder carcinoma has a decided advantage over the use of the lower voltages. It is possible to administer a tumor sterilizing dose through three comparatively small fields. With the lower voltage this tumor dose could be obtained only by a large number of fields because the skin of one single field would not tolerate the amount which it is possible to administer with supervoltage radiation. —*Mary Frances Vastine.*

Rose, D. K. Open x-ray therapy in carcinoma of the bladder. *J. Urol.*, March, 1946, *55*, 267–272.

This article describes the improvements made by the author in his technique for direct bladder irradiation for malignancy. He emphasizes the similarity between skin and bladder carcinoma and the importance of presenting a flat cancer bearing surface to the roentgen rays for even distribution of the irradiation. To this end he tries to remove redundant tissue, bloody urine and blood clots from the tumor surface to prevent absorption of the roentgen rays by this material, before they reach the tumor surface.

Rose uses pentothal sodium or any general anesthetic. In this article he includes illustrations of the abdominal retractor he employs, the marsupialization technique and introduction of a lead cuff to protect the lateral bladder walls as well as lead protection for the abdominal wall.

Results are not satisfactory in treatment of bladder outlet tumors or extensions.

Originally the author used up to 6,000 r in one exposure but the development of bowel strictures and vesicovaginal fistulas from destruction of the floor of the bladder caused him to reduce the dosage. He now employs 3,300 r in one treatment. The other factors used are not stated.—*R. M. Harvey.*

MISCELLANEOUS

Marshak, Alfred, and Byron, R. L., Jr. A method for studying healing of bone. *J. Bone & Joint Surg.*, Jan., 1945, *27*, 95–104.

A method of using radioactive tracers to study the healing of bone in vivo is described in this paper. Radiostrontium (Sr^{89}), the radioactive isotrope of strontium and radiophosphorus (P^{32}), the radioactive isotrope of phosphorus were used. Radiostrontium was chosen because, in the Periodic Table, it is closely related to calcium and behaves physiologically in a manner similar to that substance, but for technical reasons, is more convenient to use than radiocalcium (Ca^{45}). Both radiostrontium and radiophosphorus give off beta particles of approximately the same energy value, which can be measured easily with a Geiger-Müller counter. Methods were devised to determine the uptake of phosphorus and strontium by healing bone, by means of which it was possible to study the changes in the mineral metabolism from day to day, the relationship between phosphorus and strontium, and the effects of age on the healing of bone. The investigations were made in vivo in rats; with certain modifications they could be applied to studies on the healing of bone in man.

A method was devised for making uniform lesions in the bone. Determinations of the uptake by healing bone of radiophosphorus and strontium at different stages in the healing process were made. The uptake by healing bone of phosphorus and strontium was compared, and the effect of the age of the animals upon the uptake of phosphorus in the healing of bone was studied. In young animals they found that the latent period for an increased uptake of phosphorus is shorter than in older animals (two as compared with four days). The maximum uptake of phosphorus and strontium, they found, occurs on the eighth to the tenth day in young rats as compared with the maximum of fifteen days reached in old rats. This parallels the gross healing of the old and young bones. By comparing the ratio of uptake of strontium and phosphorus, it was seen that this ratio tends to increase later in the healing process; it may be possible to utilize this tendency to differentiate between the proliferating and the calcifying stages of bone healing.—*R. S. Bromer.*

Maun, Mark E., and Dunning, W. F. Is the biopsy of neoplasms dangerous? Experi-

mental study. *Surg., Gynec. & Obst.*, May, 1946, *82*, 567–572.

Clinicians are prone to avoid the incision or removal of neoplasms for fear that the diagnostic procedures may serve to disseminate tumor cells, but the reluctance to obtain a biopsy specimen for histopathologic study often delays adequate therapy and invalidates an accurate diagnosis. It is generally accepted as a truth—and there is some experimental evidence to support the belief—that the rupture of the capsule of a tumor or the destruction of adjacent tissue structures incurred in obtaining a biopsy specimen may serve to open vascular and lymphatic channels to tumor cells. Physicians, therefore, avoid manipulation of suspected tumors in order to prevent local extension or metastases. These concepts imply that the extent of neoplasms can be roughly judged by gross inspection and that tumor metastases are usually limited by the observed tissue barriers.

Summary of Experimental Work Done by the Authors. Rats bearing transplanted fibrosarcoma, adenocarcinoma, and squamous cell cancer were operated upon and compared with an unoperated upon control series for length of survival after transplantation and percentage in which metastases were detected.

The average survival period of rats bearing transplanted fibrosarcomas was significantly prolonged by excision of the tumor nine days after transplantation but the percentage of lung and lymph node metastases was not altered.

The average survival period of rats bearing transplanted adenocarcinoma was significantly prolonged by the removal of a considerable mass of the tumor sixty days after inoculation. The rats operated upon which survived the rats of the control series had a significantly higher percentage of lung metastases.

A simple biopsy performed on rats bearing transplanted adenocarcinoma did not affect the average survival period of the rats or increase the percentage with lymph node and lung metastases.

A simple biopsy of transplanted squamous cell cancer did not affect the average survival period of the rats or increase the percentage of metastasis to the lymph nodes, lungs, and skeletons.—*Mary Frances Vastine.*

Dobyns, Brown M. Studies on exophthalmos produced by thyrotropic hormone. II. Changes induced in various tissues and organs (including orbit) by thyrotropic hormone and their relationship to exophthalmos. *Surg., Gynec. & Obst.*, May, 1946, *82*, 609–617.

Tissues of animals which during life had been given various thyrotropic hormone preparations and on which very accurate records of the devlopment of exophthalmos had been made were studied grossly and by several histologic techniques.

Antituitrin T, which produced the most striking exophthalmos, was found to produce gross and microscopic changes in connective tissues not only in the orbit but elsewhere in the body.

The study showed that as a result of the administration of thyrotropic hormone there was a generalized alteration in fat deposits, which are rapidly depleted of their fat contents and are replaced by a more translucent gelatinous material. Histopathologically these changes in fat and connective tissue are characterized by the presence of edema and the infiltration of large numbers of polymorphonuclear leukocytes, lymphocytes, and tissue macrophages. Early in the reaction the polymorphonuclear leukocytes and tissue macrophages are loaded with tiny droplets of fat. Later an increase of new connective tissue is evident. This reaction was correlated with the exophthalmos which had previously developed in these animals.

Skeletal and cardiac muscle fibers during the early period of administration of thyrotropic hormone lost their striations and were found to contain multitudes of tiny fat droplets, which lined up in a pattern representing the cross-striations of the fibers. New cellular elements were present among muscle fibers. Considerable significance was attributed to a close correlation between the presence of fat in the muscles and the myasthenia present in these animals before death, a fact which may have clinical correlations.

Further evidence of alterations in fat during the early administration of thyrotropic hormone is the presence of large amounts of fat in the liver, kidneys and epithelium from several locations, as well as large phagocytic cells of the lungs, spleen and lymph nodes.

These changes took place regardless of the presence or absence of thyroid.—*Mary Frances Vastine.*